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# THE INSULIN-SHOCK AND METRAZOL TREATMENTS OF SCHIZOPHRENIA, WITH EMPHASIS ON PSYCHOLOGICAL ASPECTS

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In the past several years two new therapeutic methods have commanded the focus of psychiatric attention—the *insulin-shock* and *metrazol* treatments. The former, through large doses of insulin, effects a reduction in blood sugar and finally gives rise to definite shocks; the latter, through intravenous metrazol injections, produces epileptiform convulsions. Both methods, though leaving us in doubt as regards the actual nature of their therapeutic action, have infused new hope into the treatment of the psychoses, particularly schizophrenia. Both have definitely opened up new fields of research for the psychiatrist, biochemist, physiologist, neurologist, and psychologist. The aim of this paper, which makes no claim to exhaustiveness, is to acquaint the psychologist with these important therapies.[1]

## Insulin-Shock Treatment

*History.* The insulin-shock treatment of schizophrenia was discovered by Sakel, who came upon it through the auspicious interplay of observation, deduction, and accident. It evolved from experimentation in the treatment of morphine addiction (**44, 45**). Sakel, assuming adrenalin to be operative in producing the withdrawal symptoms characteristic of morphine addiction, employed insulin as an adrenalin antagonist. Although careful to limit the doses so as

[1] Both therapies, though employed chiefly in the treatment of schizophrenia, have been tried with other mental abnormalities. This review, however, is limited to the use of these therapies in schizophrenia, covering work up to the spring of 1939.

to produce only mild hypoglycemia, occasionally he was surprised by deeper hypoglycemic reactions than were intended, and the patient slipped into severe shock. Observing beneficial effects from such occurrences, he used hypoglycemic shocks first in treating other excited states, and then in treating actual psychoses. The results obtained were encouraging, especially in cases of schizophrenia. In 1933 he began treating a large series of cases in the psychiatric hospital associated with the University of Vienna. From here the treatment spread rapidly to all parts of the world. In 1933 [2] he reported on his new treatment of schizophrenia to the Verein für Neurologie und Psychiatrie in Vienna.

*Technique.* In presenting the technique of administering insulin-shock therapy we will describe the general method outlined by Sakel (**47**)[3]; this is the prototype from which all others derive. The technique is not rigid; Sakel is explicit on this point, stating that "the method itself is not stereotyped, and allows a wide scope of variation, so that frequent modification may often be necessary for any one patient. Success depends very much on the use of the right modification at the right time, in accordance with the changing behavior of the patient in the various phases" (p. 13).

The essence of the treatment is the repeated production, usually daily, of severe hypoglycemic shocks. These are effected through deep intramuscular injections of insulin, given in bed in the early morning fasting. Several days of cautious preliminary exploration are usually necessary to determine the shock dosage; this dosage may later be decreased in some cases to two-thirds or even a half, as sensitization takes place. Actual shock, which occurs from one to several hours after injection, is preceded by characteristic signs of hypoglycemia. These are weakness, hunger (which may be severe), perspiring, and progressive somnolence. Somnolence may be interrupted from time to time by episodes of excitement. As somnolence deepens, the patient loses contact with the surroundings and passes into shock.[4] Transitory spasms of the extremities, fine muscular twitchings, intense tremors of the entire body, stertorous breathing, and semivocalizations in the nature of grunts and snorts are common.

[2] This report was subsequently published in English (**46**).

[3] All references to Sakel's writings, unless otherwise specified, will be to his monograph (**47**).

[4] The term 'shock' has not been rigidly defined. The writers employ it in the sense suggested by Küppers (**23**), in which lack of ordinary awakability is made the criterion. Müller (**31**) employs a more concrete criterion, the inability to drink.

The body temperature may drop several degrees. As shock progresses, it may lead to deep coma. In some instances epileptiform convulsions either precede or accompany coma. After such convulsions spontaneous awakening usually takes place.[5] Coma, which is characterized by complete loss of response to any kind of stimulation, tends to occur from three to four hours after injection. It is allowed to continue for from a few minutes to an hour, depending on the psychotic picture and physical condition of the patient. Termination of shock is accomplished in several ways. If the shock is superficial and the patient's coöperation can be obtained, sugar-water is given by mouth; otherwise, a tube-feeding is administered. If a quick arousal is desired, an intravenous injection of glucose is given. This brings immediate awakening. After the patient is fully awake, regardless of method of arousal employed, a regular meal, rich in carbohydrate, is fed to prevent subsequent relapse into shock. The general sequence of events in coming out of shock is just the reverse of that in going in.

Individualization of treatment is important. By varying length, depth, and type of shock a range of reactions may be produced. Sakel strives to evoke, and then fixate, a reaction which is the diametric opposite of the psychotic picture he is trying to combat. Fixation is accomplished through properly timing the termination of shock. He states: "The observation that there is a tendency toward fixation of the clinical picture which happens to dominate at the time of termination, helps to explain the good results of the following modification . . ." (p. 53). The modifications recommended by him are these: Paranoid patients should be subjected to deep comas.[6] Stuporous patients should be aroused from shock at the point of greatest activity; if activity is insufficient, they should be subjected to metrazol convulsions. Excited catatonics should be put into deep somnolence, though not into actual coma, and convulsions should be avoided for them. No patient, other than a stuporous one, should be aroused during a reactivated psychosis (*infra*).

Improvement may be evidenced at first only by abatement of the more gross symptoms, such as hallucinations and delusions. Later, a disappearance of the more subtle emotional abnormalities may fol-

[5] The awakening is probably due to a sudden rise in blood sugar; the muscular activity stimulates adrenalin secretion, which, in turn, mobilizes liver glycogen.

[6] Comas may be encouraged by minimizing external stimulation; if necessary, they can be brought on through administration of luminal.

low. Occasionally a patient will become lucid suddenly and remain so from that time on; but such phenomenally sudden recoveries are rare. Usually lucidity is fleeting in its first appearances, and is confined to the hypoglycemic somnolent periods immediately preceding and following shock. As improvement continues, the lucid periods become progressively longer and extend further and further into the posthypoglycemic state (43). Increasing lucidity and accessibility necessitate more than ever the employment of tact in handling the patient. Sakel describes the situation as follows:

> "One got the impression that the patients were so impressionable at this stage that they responded to almost any kind of suggestion from the environment. . . Probably the most frequent reactions to environmental influences of this kind were paranoid attitudes" (p. 37).

Such periods of lucidity and accessibility afford the physician an opportunity to establish rapport (21) and to initiate psychotherapy (7).

After improvement is well under way, a temporary return of psychotic symptoms is not uncommon. Sakel describes this condition, which he calls "reactivated psychosis," as follows:

> "The symptoms which occur during hypoglycemia vary considerably in different phases of treatment. Patients who show a short period of lucidity during hypoglycemia during the early phase of treatment, only to remain psychotic for the entire remainder of the day, later show a complete reversal of reaction and remain symptom-free throughout the day, but may show a certain reactivation of their psychosis during hypoglycemia" (p. 126).

James, Freudenberg, and Cannon (21) explain the reactivated psychosis ". . . as a combination of the insulin psychosis observed in mentally normal diabetics, and the actual psychotic condition for which the patient is being treated." To the writers it appears that the combination of symptoms in hypoglycemia is of the kind found in every organic mental reaction. It consists, first of all, of symptoms which are primarily due to the noxious agent; this may be a brain trauma, an infectious process, or a toxic state such as hypoglycemia. These conditions uniformly give rise to clouded consciousness, delirious symptoms, memory disturbances, etc. In addition, symptoms of a more individual nature may appear; these are especially prominent in psychotics, and seem to account for the so-called reactivated psychosis. We have frequently observed, especially in catatonics, that the same patterns which had previously been present during the severe acute psychosis reappeared in the clouded condition of hypoglycemia. These patterns may continue with remarkable

uniformity, as Palisa (35) points out. In different individuals, however, the shock picture varies widely.

Length of treatment, *i.e.* the number of insulin shocks given, varies according to improvement shown. Treatment should be continued, if possible, until all psychotic symptoms disappear. Two or three months may perhaps be considered a representative course. The question of how long to continue treatment in the absence of improvement is up to the physician. Müller (32) urges a trial of at least three months. Termination of treatment is not brought about abruptly; instead, milder, nonshock doses of insulin are given for a few days to prepare the system for discontinuance of exogenous insulin.

*Dangers.* The dangers of insulin-shock treatment should neither be exaggerated nor ignored. Statistics on fatality rates range between .5 and 1.5% (26, 32). For the most part, fatalities have occurred during unintentional protracted shocks—that is, when glucose or other agents fail to bring awakening. Organic brain syndromes have been reported as sequelae to such protracted shocks (13, 39); most of these, however, proved to be transitory. The dangers of the treatment may be decreased by first giving a thorough physical examination, including an electrocardiogram. Patients showing signs of myocardial damage, tuberculosis, and severe arteriosclerosis should be excluded from therapy.

*Results.* Adequate appraisal of the therapy is as yet impossible. Truly objective indices are not available. Uniform bases of diagnosis and precise criteria of improvement are lacking. Actually comparable control groups have not been used. Finally, the therapy is too new to permit determinations of the permanency of remissions ascribed to it. Despite these limitations to evaluation, the widespread use of the treatment with but few dissenting reports is suggestive of its effectiveness. As Freudenberg states (14): "All authors who have written on the subject, with a few exceptions, regard insulin therapy as an important step forward in the treatment of schizophrenia." In this review we can but cite a few of the more substantial statistics in point, with the hope that they are representative.

Sakel, reporting on his original group of approximately 50 insulin-treated cases, indicates full remissions (apparent cures) in 70% and social remissions in an additional 18%. His figures are considerably larger than those reported by other investigators. The discrepancy is due, in part, to the fact that his cases, being of more recent onset, presented a more favorable prognosis. More representative are the

figures of Müller (32) and Malzberg (26), who have carried out extensive surveys. Müller's figures are based on the combined data from 22 Swiss institutions and comprise 495 treated cases. He reports full or social remissions (patients allowed to return to their occupations in apparent good health), according to duration of psychosis before treatment, as follows: up to six months, 59%; six to twelve months, 52%; one to two years, 27%; over two years, 11%. Malzberg's figures are based on the combined data from 19 New York State hospitals and comprise 1039 insulin-treated cases and 1039 controls. His controls were matched with the treated cases for institution, sex, and type of dementia praecox. He reports 13% recovery and an additional 54% improvement in the treated cases, as compared with 3.5% recovery and 19% improvement in the control group. These data, when presented according to duration of psychosis before treatment, reveal the following percentages of recovery (mere improvement not included): *treated group*—up to one month, 43%; one to three months, 33%; seven to twelve months, 16%; one to two years, 12%; and three to five years, 4%; *control group*—up to one month, 8%; one to three months, 7%; seven to twelve months, 0%; one to two years, .6%; and three to five years, 0%. In comparing these figures with those of Müller it must be borne in mind that Müller's data include social, as well as full, remissions. Both Müller and Malzberg, as well as Humbert and Friedemann (20), report more favorable prognosis under insulin treatment for the catatonic and paranoid types than for the hebephrenic type. Malzberg reports the same trend, though with generally lower remission rates, for untreated cases. Müller finds the more favorable prognosis of paranoids under treatment to hold only for cases of less than a year's standing, and the prognosis of catatonics better in excited than in stuporous cases.

Not all recoveries or improvements are permanent. Relapses are reported by Malzberg in 24.5%, and by Müller in from 6 to 13%, of cases in remission. Müller finds the higher relapse rates in cases of long standing.

*Neurological Aspects.* Abnormal neurological symptoms, of varying degrees of severity, always accompany deep insulin shock. Sakel describes the general neurological change essentially as follows: During mere somnolence the reflexes remain intact, but with deepening shock they become pathological. Babinski, Oppenheim, and Mendel-Bechterew reflexes appear. Brief transitory muscle spasms, myoclonic twitchings, and intense tremors of the entire body may be

observed. If very deep coma ensues, all of the reflexes, including even the corneal and swallowing reflexes, disappear, leaving the patient in an areflexic state, with complete atonia of the entire musculature. Epileptiform convulsions are not infrequent. As the patient gradually awakens from shock following tube-feeding, the reflexes return in the reverse order.

Angyal (1) attributes the sequential nature of the neurological phenomena to a progressive impairment of cerebral function. He conceives this impairment as starting in the frontal poles, proceeding to the motor areas, and finally descending to the brain stem and medulla. His conception is in harmony with the theory that the phylogenetically recent structures of the brain are the more vulnerable and are the first to show disturbances of function. Pap (**36**), observing choreiform and athetosic movements and decerebrate rigidity, emphasizes the extrapyramidal nature of the shock motor symptoms. Our own observations indicate involvement of both cortical-pyramidal and extrapyramidal structures and suggest less regularity in the sequence of neurological symptoms than the foregoing descriptions might imply. Sakel and others emphasize their observations that the dominant hemisphere is affected first and recovers last in hypoglycemia. We doubt that this is a universal tendency. Orenstein and Schilder (**34**) cite evidence to the contrary, and our own observations have not indicated such a tendency. Incidentally, the most severe neurological syndrome we have seen, a hemiplegia of two hours duration, contradicted it; this hemiplegia involved the left side of the body in a right-handed individual.

Changes in the activity of the autonomic nervous system are common. These changes consist in shifts of tone from the parasympathetic to the sympathetic division, and vice versa (**50, 54**). The shifts appear to correlate with emotional state; fear and excitement are accompanied by dominant sympathetic tone. Increased sympathetic tone seems to be a characteristic forerunner of epileptiform convulsions in insulin shock. We regularly observe an increased blood pressure and pulse rate, and maximally dilated pupils a few minutes before seizure. In this latter respect, in our opinion, the insulin convulsion differs from the metrazol, in which maximal dilatation does not occur until the end of the tonic spasm *(infra)*.

Electroencephalographic studies have shown both the presence of abnormal brain waves in schizophrenics and the dissolution of these waves with insulin treatment. Lemere (**25**) and Hoagland, Rubin, and Cameron (**19**) report the presence of excessive delta waves in

the electroencephalograms of schizophrenics; these waves disappeared after insulin treatment. Lemere also reports a strengthening of alpha rhythm during treatment; the alpha rhythm reverted to its pretreatment level, however, within a few weeks after insulin discontinuance.

*Psychological Aspects.* Interesting psychological abnormalities accompany hypoglycemia, especially the preshock and awakening phases. In some respects they resemble, and in others differ from, abnormal phenomena accompanying surgical anesthesia and other delirious states. Some of these phenomena are externally observable; others can only be arrived at through subjective report. Not all are to be found in any one case, as individual differences in reaction to hypoglycemia are great.

We shall report a few of our own observations. The effect of insulin on the emotional state varies widely from patient to patient. Furthermore, the emotional reaction does not necessarily correlate with emotional state preceding injection. Some patients, showing signs of fear before insulin, may remain apprehensive throughout the whole hypoglycemic period; others, showing the same signs, soon become quiet and relaxed under the effect of insulin; still others, at ease before injection, become almost frantic as hypoglycemia develops. One patient, who always resolved to rest quietly during hypoglycemia, became agitated, sat up in bed, and threw her arms into the air like a drowning person, just before going into shock. This was a daily occurrence with her. She described the experience of going into shock as a feeling of "submerging" or "being overwhelmed." It is apparent that the experience itself of going into shock produces fear in some cases.

The manner of going into shock varies. Some patients simply fall asleep and pass directly into shock from this state. More often, however, a state of clouded consciousness, lasting from a few minutes to an hour or more, precedes the shock. A similar, but shorter, state accompanies awakening. The clouding at times may be so slight as not to be readily apparent. Not infrequently we have conversed with patients in hypoglycemia who seemed completely lucid; yet on returning to them five minutes or so later, and finding them apparently unchanged, we found them oblivious of the previous visit. Such experiences indicate an impairment of retentiveness for new impressions, an anterograde or continuous amnesic state. As clouding progresses, the association processes become sluggish, retardation of speech appears, general reaction time increases, and verbal or motor

perseveration may occur; finally, the patient passes into a severe psychomotor retardation tantamount to a stupor.

It seems characteristic of this clouded, stuporous state that perception and comprehension are impaired earlier than speech and expression in general. Patients showing no indication of comprehending simple questions will later surprise one by accurately reproducing them, explaining that they understood them originally, but had been unable to speak. Such phenomena resemble motor aphasias; we doubt, however, that motor aphasia, unless conceived in a very broad sense, is the correct term for this verbal unresponsiveness. The impairment is not limited to speech, but involves all motor spheres; no attempt is made to substitute either writing or gestures for speech. The experiments of Küppers (**24**) furnish evidence in point. He induced three assistants to undergo hypoglycemic shocks. The number of shocks for the three totaled 36, several of which were allowed to proceed right up to the point of coma. The subjects reported the following general experiences: sensations of hunger and exhaustion; listlessness and distaste for activity, coupled with irritability in case rest was disturbed; and ideational paucity with impaired conceptual ability. More significant for the question at hand, however, is Küppers' observation that they failed to respond to painful stimulation (pinching) by any sort of defense movement. This is important because, after coming out of coma, the subjects not only remembered the stimulation, but described it as being quite painful. Küppers points out that with respect to pain sensitivity, these findings indicate a dissimilarity between the hypoglycemic stupor and the delirium preceding narcosis.

Perceptual anomalies are common. These embrace spatial, temporal, and motional distortions, and distortions of vividness and intensity. Spatial anomalies are reported by Benedek (**6**). They include distorted depth perception, in which persons and objects appeared to possess but the two dimensions of height and breadth, and micropsic and macropsic distortions of size. Temporal distortions are reported by Benedek (**6**) and Pisk (**38**). Several of Benedek's patients related experiencing a slowing up in the flow of time (present). More commonly, however, they erred in estimating past time. One patient estimated 15 minutes as being several hours, and reported that during this time he had relived all his life experiences. A patient of Pisk, on the other hand, perceived everything in the environment as speeded up; music seemed to her to be played much faster than it actually was, and patients and nurses appeared

to walk more rapidly than usual. Distortions in the perception of motion are mentioned by Benedek (6). The ability to merge the sequence of movements into a meaningful whole was diminished; normal movements seemed jerky, or else the moving object appeared to occupy several positions simultaneously. The patient describing these experiences likened them to some of the earliest movies in which the illusion of motion was imperfect. Anomalies in which sounds seemed louder, colors more vivid, and contours sharper than usual also are reported by Benedek (6). It should be mentioned that patients experiencing these perceptual distortions usually realize that they are due to the insulin. The above-described perceptual anomalies resemble those characteristic of hashish and mescaline.

Aphasic and apraxic phenomena are encountered. They are more common to the awakening than to the preshock phase. Palisa (35) and Sakel describe the gradual return of speech during awakening. The first signs are frequently the utterances of groans and other primitive sounds. Then come inarticulate babblings, which may be followed by a kind of baby talk. This latter has been considered a manifestation of an ontological regression (47); other similar manifestations are thumb-sucking, reporting one's age as of childhood, and mistaking the physician for one's childhood physician. Slightly later, a transitional phase of sensory and motor aphasia may occur; here, ungrammatical speech and literal paraphasia are the prominent features. The truly aphasic nature of these phenomena, which comes out during clinical examination, is also indicated by the patients' awareness of their expressive difficulties. A patient of Palisa (35), a student with medical training, referred to the condition expressly as motor aphasia. Another of his patients reported the experience in these words: "I don't know—I wanted to talk but always swallowed—the letters—the words—I couldn't get them out—I had them, though—talking turned into swallowing." The aphasic disturbances associated with shock, though most interesting from a psychological viewpoint, are difficult to study because of their transitoriness; they change from moment to moment and are usually accompanied by clouded consciousness. Occasionally, however, aphasias, and apraxias too, will continue well into the waking state. In two of our own patients a clear-cut motor aphasia persisted for several hours after consciousness had cleared. Only verbal expression, and not comprehension, was impaired. In addition to the motor symptoms, an amnesic aphasia persisted for some time. One of the patients referred to a pencil as "a thing to write." In a third patient we observed, in

combination with a slight motor aphasia, a condition which might be termed *swallowing apraxia*. This condition persisted for about an hour after she had fully regained consciousness. Several times she tried to drink; but although she tipped her head back and made all sorts of mouth and tongue movements, swallowing did not occur. Since she was not at all resistive, commented on her inability to swallow, and swallowed reflexly when tactually stimulated on the pharynx by a tongue-depressor, it appears that her difficulty lay in the volitional initiation of the swallowing act. We believe that these swallowing apraxias are fairly common, but because of their fleetingness and the clouded condition of the patient when they occur, they usually cannot be demonstrated as apraxias.

Amnesic phenomena, some of which have already been mentioned, occur. They include complete amnesia for deep shock and impaired retentiveness for impressions in the preshock and awakening phases. These are to be expected, of course, because of the clouded condition of the patient at such times. Retrograde amnesias for periods of varying lengths may be present immediately following shock; usually, however, they clear up later.

Various reactions which appear to be manifestations of disinhibition are observed. Some of these resemble phenomena common to alcoholic and other toxic conditions. In three male patients we observed a state of uncontrollable excitement developing in the preshock phase a couple of hours after injection. The state was strikingly similar in all three individuals, and in each individual occurred day after day with almost photographic uniformity. It lasted for about 20 minutes, during which several persons were required to hold the patient in bed. One patient exclaimed to his nurse: "Take off your glasses, I must hit you!"; and another cried in response to the physician's attempt to quiet him: "Doctor, I can't stop it—I can't." These states disappeared when the onset of shock was hastened by increasing the insulin dosage, but returned again in one patient whose dosage had to be decreased to prevent convulsions. They were characterized by clear consciousness; subsequent recollection of happenings was good. Fear symptoms were absent, and there appeared to be no paranoid basis for the states. Rather, they seemed to be simply an enormous, uncontrollable motor discharge. The patients later described their subjective experience as that of having lost control of their actions. The detached, impersonal nature of these excited states, the patient's awareness of the futility and undirectedness of his behavior, differentiates them from epileptic

twilight states and alcoholic and other toxic psychoses. In this respect this type of hypoglycemic excitement more closely resembles the organic 'drivenness' found in certain diseases of the subcortical ganglia, such as epidemic encephalitis. Quite likely temporary changes in the subcortical ganglia are involved in the above-described hypoglycemic episodes.

Other types of impulse release have also been reported. Palisa (35) observed in the awakening phase in a number of cases a sort of frank eroticism which did not, however, extend to gross sexuality; rather, it was a bid for affection. Palisa comments that this was in direct contrast to the patient's usual schizophrenic seclusiveness. Orenstein and Schilder (34) report the appearance of hidden sexual material, *i.e.* homosexual transference. Palisa (35) also mentions, in connection with about this same phase of the awakening period, the sudden appearance and disappearance of aggressive attitudes toward the environment.

A feeling of relaxation, accompanied by hypomanic attitudes, often follows awakening from shock (34). It is our impression that such attitudes are a favorable sign, as we have encountered them especially in cases showing improvement. Furthermore, we have observed a phase of slight elation, lasting from several days to weeks, following termination of treatment in recovered cases.

Recent findings suggest possible prognostic value for certain psychological tests in insulin-shock treatment. Bolles, Rosen, and Landis (8) report indicative results from a battery of performance tests. These tests, designed to measure the capacity for "abstract" behavior, had previously been found to show characteristic differences between normal subjects and schizophrenic patients. After applying the tests to a series of 19 schizophrenic patients prior to insulin therapy, Bolles, Rosen, and Landis compared the test results with the subsequent outcomes of treatment. They summarize their findings as follows: "Those patients whose performances were very poor on these tests tended to show no improvement under insulin therapy. Among those patients whose performances were relatively superior, the number of improvements was larger." Similarly, Piotrowski (37), on the basis of 25 pretreatment records, finds suggestions of prognostic value in some of the Rorschach indices. Among those indices on which a high score seemed suggestive of favorable prognosis were whole responses, human movement interpretations, and color responses; among those indices in which a low score appeared to suggest a favorable prognosis were rare detail responses.

## METRAZOL TREATMENT

*History.* The metrazol[7] convulsion therapy for schizophrenics was discovered by Meduna. The therapy is based on the assumption of a mutual antagonism between the schizophrenic process and epilepsy. Several reports suggested such an antagonism: It had been observed that when epilepsy is complicated with a schizophrenic syndrome, convulsions become rare or cease entirely (**33**); cases of catatonia had been known to recover after epileptiform convulsions (**30**); typical epileptiform convulsions are rare in schizophrenics (**51**). Meduna, reasoning from these reports, deliberately produced epileptiform convulsions in schizophrenics. Originally he employed camphor as a convulsant; soon, however, he abandoned it for metrazol, which produced a more immediate reaction. He first published on the treatment in 1935 (**27**).

*Technique.* The technique of metrazol treatment is relatively simple (**22, 28, 41**). The convulsion is evoked in bed. Attendants, in addition to the physician, should be present to protect the patient. Treatment is begun with an initial dose of from 3 to 5 cc.'s of 10% metrazol solution, the dosage depending on the sex and general condition of the patient. Women usually require less than men. Frequently the dosage has to be increased later, as a tolerance for the drug is built up. The metrazol is given rapidly by intravenous injection. Within a few seconds the patient coughs, becomes bewildered, restless, fearful, and confused, and then goes into convulsion. During the convulsion he is unconscious. The seizure, which is of the epileptiform type, lasts from about 30 to 80 seconds. Tongue-bites are prevented by inserting a gag between the teeth; this can easily be done, as wide opening of the mouth invariably occurs during the tonic phase of the seizure. After the convulsion the patient usually remains comatose for several minutes; then, either immediately or after an intermediate twilight state of clouded restlessness, he falls asleep. Convulsions are induced two or three times a week. A course of treatment comprises from 12 to 30 seizures, or, in terms of time, from 4 to 15 weeks. Meduna (**28**) urges continuing treatment through three additional convulsions after recovery, so as to stabilize the effect.

*Neurological Aspects.* The sequence of neurological events occurring during the metrazol convulsion has been carefully analyzed by Strauss, Landis, and Hunt (**52**) by means of ultrarapid cinematography and electromyography. They found that the convulsion, last-

[7] Known in Europe as cardiazol, and chemically as pentamethylenetetrazol.

ing from 45 to 60 seconds, consisted of three distinct, though somewhat overlapping, stages: first clonic, tonic, and second clonic. The first stage, with an average duration of 10 seconds, consists in the alternating contraction of agonistic and antagonistic muscle groups at the rate of two to four two-phase movements a second. The tonic stage, with an average duration of 5 to 10 seconds, is characterized by simultaneous high potentials in all muscles. The second clonic stage, with an average duration of 30 to 40 seconds, consisting in the alternation between simultaneous contraction and relaxation of all muscle groups, represents an alternating disappearance and reappearance of the continuous innervation of the tonic stage. The alternation is rapid at the beginning (12 to 14 contractions a second), gradually slowing to one or two a second towards the end, and terminating in complete relaxation. Careful analysis reveals definite differences between the two clonic stages. In the first, adduction and inward rotation of the limbs is associated with flexion of the knee, elbow, and head; in the second, adduction and inward rotation of the limbs is associated with extension of knee, elbow, and head. Furthermore, amplitudes of movement are greater in the first stage. These authors, on the basis of their findings, conclude that the first clonic stage is probably the expression of cortical stimulation, while the second is not. The second appears to be a state of repeated interruption of the decerebrate rigidity which was present in the antecedent tonic stage. The tonic stage seems to be one of continuous decerebrate rigidity. The fact that arm flexion, even up to 90°, sometimes occurs does not contradict this stage; decerebrate rigidity in humans is frequently accompanied by arm flexion.

Angyal (**2**) regards the first clonic stage as a psychomotor condition. The writers, while in thorough agreement as to the presence of psychomotor agitation after injection, regard this agitation as part of the aura rather than of the convulsion proper. Individual convulsion patterns apparently vary considerably. Meduna (**28**) reports tonic stages ranging from 7 to 26 seconds, and makes no mention of the first clonic stage. We have observed extremely short first clonic stages, as well as relatively long tonic stages, in certain cases. Sometimes the tonic stage begins with torsion of the head and body to one side.

Smooth, as well as striated, muscles are involved in the convulsion. Emptying of the bladder and ejaculation without erection in the tonic stage, and defecation at the end of the second clonic, are not uncommon (**28**). Severe blanching of the face, a vasoconstriction

manifestation, is often the first significant sign after injection. Sometimes, however, excessive reddening of the head and trunk, which may be accompanied by the appearance of goose flesh, occurs instead. Later, during the second clonic phase, cyanosis predominates. Meduna (**28**) attributes the cyanosis to impaired breathing in the preceding tonic stage. Pulse and blood pressure, which can only be measured during certain parts of the seizure, show characteristic changes. Shortly after injection, pulse rate may increase. It becomes still more rapid after the convulsion is over. Blood pressure is raised. Meduna (**28**) reports rises of from 5 to 15 mm. Hg in diastolic, and from 20 to 30 mm. in systolic, pressure immediately after the seizure. The pupils also undergo changes; in fact, the metrazol seizure affords an unusual opportunity for studying pupillary reactions in the epileptiform type of convulsion. Meduna (**28**) asserts that the pupils are usually dilated maximally and that the pupillary reflex is invariably absent.

While Meduna's description holds roughly, we agree with Georgi and Strauss (**16**) that it is not completely adequate. They report wide, fixed dilatation during the tonic and clonic stages, followed immediately by contraction, with continued unresponsiveness to light, after the first inspiration. They also report a hippus, *i.e.* a fluctuation consisting in rapid alternate dilatation and contraction of the pupils, occurring before recovery to normalcy. Our own observations indicate the following general picture, from which, of course, deviations often occur. Often the pupils are dilated before injection, presumably because of fear; however, if they are narrow before injection, they ordinarily dilate slightly before the onset of seizure, usually at the moment of coughing. With the onset of convulsion they suddenly dilate further, but do not reach a maximum until the middle of the tonic stage. From this point on they fail to react to light. With the first deep breath after the convulsion they often contract suddenly, but only for a few seconds; then either a hippus, or successive contractions and dilatations, each lasting several seconds, may occur before normalcy is reached. These observations lead us to the conclusion that the pupillary changes during the metrazol convulsion are the expression of sympathetic-parasympathetic imbalance, and that the so-called fixed pupil is essentially a 'pupillotony,' as it is in all epileptiform convulsions.

During the recovery period following seizure, changes in the reflexes, both deep and superficial, are present (**28**, **49**, **52**). A completely flaccid state usually follows the convulsion. In this state the

Babinski reflex cannot be elicited; it appears later, however, while the patient is still unconscious, and persists for several minutes (49). Foot clonus is established somewhat earlier. In our experience, the most persistent pyramidal sign following the seizure has been the absence of Mayer's reflex (adduction of the thumb as a result of passive flexion of the first phalanx of the fourth finger). Frequently, the abdominal reflexes are missing for several minutes, and during this otherwise flaccid state, the teeth remain tightly clenched. The clenching persists for some minutes, during which the mouth gag cannot be removed except by force.

If the dosage is insufficient, as is sometimes the case since tolerance for metrazol increases during treatment, inadequate reactions result. Angyal and Gyárfás (3) distinguish two degrees of such inadequate reactions, psychic and abortive-motor. The former consists of vasomotor phenomena which are usually accompanied by severe feelings of anxiety; the latter consists of a few clonic twitchings, or, as we have sometimes observed, a short tonic stage, in addition to the vasomotor reactions and anxiety. Both forms are undesirable in our opinion. They tend to augment the fear of treatment and are often followed by extended twilight conditions and extreme agitation. Only in rare instances have we observed improvement, instead of setback, following them.

*Mechanism of the Convulsion.* The mechanism of the metrazol convulsion remains obscure. Speculation concerning it, however, is not wanting. Meduna (28), assuming a practically identical effect for metrazol and camphor, quotes Hildebrand on the effect of the latter drug as follows: "Besides the brain cortex the autonomic centers in the diencephalon are strongly stimulated." Denyssen and Watterson (12) consider the chief effect of the metrazol to be vasoconstriction. Experimenting on 32 patients undergoing metrazol treatment, they administered amyl nitrite and histamine in conjunction with the metrazol. These drugs, which had a vasodilatory effect, opposed the action of the metrazol and in some cases prevented the convulsion. On the basis of their findings they conclude that metrazol acts solely through the vasomotor center and that the convulsion is due to a vascular spasm. Their findings and interpretation support the widely entertained view that epileptiform convulsions are the immediate effect of brain anoxemia, the anoxemia resulting from vascular spasm which impedes cerebral circulation. If it be granted that the convulsion is mediated through vasoconstriction, there still remains the problem of how the metrazol reacts on the vasomotor

center. One would suppose that transmission is through the blood stream. Yet, we doubt that this is the actual mechanism of transmission because of the short latency of the convulsion in some cases. We have frequently observed convulsions occurring eight seconds, and in some instances seven seconds, after the beginning of the injection, and Barnacle (4) reports convulsions as soon as five seconds after injection. Such short latencies would hardly allow time for the blood to flow from the vein in the arm to the vasomotor center in the medulla. Mediations through various autonomic reflexes suggest themselves as more likely possibilities. Animal experimentation seems to hold the key to the solution of the problem, an answer to which should cast new light on the complicated question of the epileptiform mechanism, especially in cases of reflex epilepsy.

*Psychological Aspects.* The psychological accompaniments of the metrazol convulsion may be grouped conveniently into those associated with the aura, or preconvulsive state, and those of the postconvulsive state, the patient being unconscious during the convulsion itself. The most commonly reported and most striking feature of the aura is a feeling of alarm, manifest in the facial expressions, spontaneous outcries, and subsequent accounts of the patients. A patient of Schilder (49) always exclaimed: "Mother, mother, help me." Such utterances seem to be more common when the onset of convulsion is delayed. The subsequent descriptions of the aura by patients vary considerably, but many indicate anxiety and a life threat. Schilder's patients described the first signs of the aura as a feeling of "electricity playing all over their bodies," or as an impression that the room was "filled with dazzling white light." Others have described the aura as an "infernal chill," "sinking slowly into a hole," "the anxiety of feeling one's self going" (20). Several of our patients reported vertigo in which the room seemed to revolve, and one of them thought the walls were coming down on her. On the other hand, Gillespie (17), who underwent a single metrazol convulsion for the purpose of self-observation, describes a somewhat milder experience:

> "After the injection of 10 c.cm. of a ten per cent solution I felt my body and limbs coming to lie straight in the bed and my head turning to a central position. Then very rapidly something seemed to move in my arms and thorax and to pass up to the base of my neck, where it stopped and increased in intensity. This sensation was not painful, but felt very clear and circumscribed, half-way between thought and sensation like the ideas in delirium. Unconsciousness developed very quickly but otherwise its onset felt like the coming of normal sleep and was not resisted."

Our impression is that fear of treatment increases after several convulsions, and that abortive seizures, in which consciousness is not lost, excite more intense fear than typical epileptiform ones. The intense emotional experience of the aura gives rise, in many instances, to severe fear of injection. In several improving patients we observed a transitory reappearance of psychotic behavior patterns during this preinjection-fear state. With increasing improvement fear still remains, though it may be better controlled. In fact, even after patients fully appreciate the beneficial effect of the treatment, they tend to continue the course only with reluctance. On the other hand, a few emotionally deteriorated schizophrenics do not seem to mind treatment at all. One of our patients of this type actually seemed to look forward to the injections and, although he showed no improvement at all, declared in a stereotyped manner after each treatment that he felt much better. The emotional attitude of patients towards metrazol contrasts with that towards insulin. Many of our improving insulin patients urged continuance of treatment.

The postconvulsive state is characterized by clouded consciousness and retarded psychomotor activity. This state, so far as we can tell, is similar to postconvulsive twilight states following other types of epileptic fit. It does not follow immediately upon termination of the convulsion, but is preceded by a coma of from a fraction of a minute to several minutes in duration, and often by a few minutes of sleep in addition. If sleep follows the coma, the ensuing clouded state tends to be less pronounced. This clouded state presents characteristic symptoms which become apparent on probing. Speech expression and comprehension are impaired. Fully as striking are amnesic aphasias in which difficulty is experienced in naming recognized objects. Schilder (**49**) maintains that in most cases patients have greater difficulty in naming parts of their own bodies than in naming other objects. Retrograde amnesia for the injection, as well as for events preceding it, is characteristic. This amnesia usually clears up, however, and memory of both the injection and aura returns. Gillespie (**17**) describes his own experience as follows:

> "On awakening I had a mild occipital headache, some aching of the mandibular joints and was conscious of a pungent smell at the back of my nose. At first I could remember almost nothing of the morning's events including the injection, and even the day before seemed indistinct, but otherwise feeling and comprehension seemed normal. Remembrance of the injection returned within a few minutes and of the other happenings of the morning more gradually, but even ten hours afterwards there was

some retrograde amnesia. For the remainder of the day the feeling was distinctly unpleasant, with headache, anorexia and malaise."

Cohen (**10**) asserts that after the convulsion the cognitive functions return in a definite order, which is as follows: esthesia, prosexia, gnosia, praxia, and mnesia. He finds that immediately after the convulsion pinpricks are not responded to; shortly after this they are responded to, but only by indefinite, mass movement. As time goes on, responses to pinpricks become more localized; they pass progressively through the stages of withdrawal of the pricked limb, scratching of a broad area contiguous to the point stimulated, and, finally, localized scratching of the exact point. During the period of inexact tactual localization he found corresponding inexact localization in the auditory and visual spheres. Addressed by name, patients turned their heads, but not necessarily towards the sound source; presented with objects they appeared to look " through " rather than at them. After the other symptoms had disappeared, amnesia, both retrograde and anterograde, was still evident.

Wittman (**56**) has studied the course of the metrazol postconvulsive confusion psychometrically. Using a battery of tests of attention, rote memory, retention, substitution, and paired associates available in different forms, she tested each of 30 patients before the convulsion and again at intervals ranging from one-half hour to 48 hours following it. Average scores indicated extreme inefficiency a half hour after the convulsion, with recovery to the preconvulsive level still not complete at the end of four hours. In a second part of the study she tested patients 24 hours after each convulsion with a view to determining rate of improvement during the course of treatment. Individual variations were noted, but average scores indicated a gradual improvement up through the ninth treatment, which improvement she attributes to increased interest, attention, and coöperation. Data beyond the ninth treatment were open to ambiguous interpretation.

*Results.* The metrazol therapy presents the same difficulties of evaluation as the insulin. We can but present a few statistics with the hope that they are representative. Meduna (**28**), reporting on the first 110 treated schizophrenic cases, indicates remissions in 54 (49%). Analyzed according to duration of psychosis prior to treatment, his data reveal the following percentages of remission: up to six months, 92%; six to twelve months, 86%; one year to one and a half years, 43%; one and a half to two years, 55%; two to three years, 25%; three to five years, 27%; over five years, 0%. Analyzed

according to clinical type, the percentages of remission are: simple type, 51%; paranoids, 55%; catatonics, 30%; hebephrenics, 67%. Beckenstein (5) presents the following results for 218 cases treated at the Brooklyn State Hospital: less than six months duration—13% recovered, 35% much improved, 44% improved, 9% unimproved; six months to two years—1.4% recovered, 15% much improved, 43% improved, 40% unimproved; over two years—.8% recovered, 5% much improved, 29% improved, 65% unimproved. He claims best results in paranoids of recent onset and poorest in hebephrenics regardless of recency of onset. Meduna and Friedman (29) report the combined results from 2937 cases treated in American and European hospitals. The gross American data indicate 19.86% full remissions and 38.43% improvement. Analyzed according to duration they reveal the following: less than six months, 60.95% complete remission and 20% improvement; six months to one year, 36.82% complete remission and 23.13% improvement; over one year, 8.36% complete remission and 37.76% improvement. The gross European data indicate 30.37% full remission. Analyzed according to duration they show 49.66% full remission for cases under, and 13.32% for cases over, 18 months. Mortality for the total 2937 cases is given as .29%.

*Dangers.* Meduna (28) makes the claim that the metrazol treatment is free from danger. He bases his contention on the fact that his data include no deaths, and that out of a thousand convulsions the only deleterious effects, aside from occasional backaches in which no injuries to the ligaments were found, were two arm dislocations and a dislocation of the jaw. Others report more serious consequences. Deaths during treatment are reported in from 0 to 2.2% of cases (3, 22, 28, 29, 42). The reported probable complications leading to deaths include pneumonia, lung abscess, and pulmonary embolism. Other complications include dislocations and fractures (29). A vertebral fracture was detected by Wespi (55), who pointed out the necessity of paying greater attention to complaints of backache. Nevertheless, fractures of spinal vertebrae were considered rare occurrences until very recently, when their incidence was investigated by Polatin, Friedman, Harris, and Horwitz (40). These investigators, through routine X-ray examinations, found a high percentage of compression fractures in the thoracic spine. They report fractures—some of them slight—in 22 out of 51 patients (43.1%) and state that the degree of injury usually bore no relation

to intensity of backache. Such findings may prove a serious objection to the use of metrazol, unless attempts to avoid this complication are successful.

### Comparison of Insulin and Metrazol Therapies

Many workers in the field of shock therapy point out that the metrazol therapy is the easier to manage because the individual treatments are less time-consuming and require less nursing care (**53**). A metrazol treatment can be completed in half an hour, with little attendant danger of postconvulsive complications. An insulin treatment, on the other hand, not only consumes several hours, during every minute of which the patient must be carefully watched, but also necessitates constant observation throughout the day because of the danger of after-shock. A more important consideration, however, is the question of the respective therapeutic values of the two treatments. At present, no absolute statements can be made on this point. Both are of value. Cases refractory to one may respond to the other, and vice versa (**29**). For catatonic cases convulsions seem to be especially helpful (**47**). Our own impression—and this is supported by the two large surveys presenting together results from both therapies (**42, 53**)—is that, in general, the insulin-shock treatment is the more efficacious.

### Combined Treatment

In an effort to retain the desirable features of both treatments, while eliminating some of the undesirable ones, Georgi (**15**) and Georgi and Strauss (**16**) have developed a combined insulin-metrazol therapy. The usual dose of insulin is given, and a metrazol convulsion is induced about an hour and a half later. Hypoglycemia is then terminated. Termination can usually be accomplished, in such instances, without tube-feeding, as the patient usually awakens after the convulsion and can be induced to drink the sugar solution. Georgi and Strauss (**16**) advise either giving insulin and metrazol together two or three times a week or giving insulin daily, with metrazol in addition on alternate days. They recommend the former for cases responding with violent seizures. Braunmühl (**9**) recommends, for schizophrenics of recent onset, starting out with insulin alone and, later, after about 20 shocks, adding a limited number of metrazol convulsions if necessary. The combination type of therapy offers the following advantages: It obviates the fear reaction so

common in the unmitigated metrazol therapy. Presumably, the somnolent state induced by the insulin tends to eliminate the fear-evoking experiences associated with the metrazol injection. The combined method lowers the threshold for convulsions, thus enabling an appreciably smaller metrazol dosage to be given. Finally, the combination therapy, in employing both insulin and metrazol, reaches cases which might be refractory to either one alone.

We are convinced that in many cases a combination of the two treatments is advisable. Such combined treatment, however, can hardly be prescribed in a blanket manner, as convulsions may have undesirable, as well as desirable, effects. Regulation should be based on the patient's reaction to a trial convulsion.

## Theory

Speculation concerning the actual nature of the mechanisms by means of which the pharmacological shock treatments effect cures has not been wanting. Much of it is vague. Each of the hypotheses tends to reflect its author's viewpoint toward the etiology of schizophrenic syndromes. Hypotheses range from the essentially physiological or organic to the essentially psychological. We can but mention a few. Among the physiological theories of insulin effect are those of Sakel, Demole, Georgi, and Freudenberg. Sakel (46) suggests the possibility of a general lowering of neural thresholds in psychotics through adrenal activity, which revives phylogenetically ancient and infantile pathway patterns latent in normal waking thought. Insulin, being an adrenalin antagonist, is supposed to reverse the process. A more definite and experimentally verifiable hypothesis is offered by Demole (11). He points to a disturbance in cerebral respiration preventing the oxygenation of the toxic products of metabolism, which accumulate and give rise to the schizophrenic symptoms. He suggests that insulin stimulates the oxybiotic processes necessary for detoxication. Georgi (15), on the assumption that schizophrenia is characterized by relative impermeability of cell membranes, suggests that insulin stimulates the exchange between the cells and their surroundings. Freudenberg (14) combines the hypotheses of Demole and Georgi into the following:

> "In schizophrenia there is probably a primary disturbance in cerebral respiration, perhaps due to some lack of oxygenating substances. This disturbance leads to a collection of toxic products, probably originating from the protein metabolism. Insulin therapy induces the oxybiotic

processes necessary for detoxication and also an irritation of the cell membranes, which results in an increased exchange between the cells and their surroundings."

Among the psychological hypotheses are those of Glueck and Schatner. Glueck (**18**) considers the various emotional and motor phenomena occurring during hypoglycemia as constituting an emotional catharsis and states that the entire situation might be viewed ". . . as a highly condensed recapitulation of the process by means of which the fate of the ego-dystonic impulses is determined, a process which takes place normally, in the case of ontogenetic development, and artificially, in the course of psychoanalytic therapy." Schatner (**48**) believes the efficacy of the insulin-shock treatment to lie in its rendering the patient sick, helpless, and dependent. This state predisposes him to a transference in which the physician becomes the mother surrogate and, as such, can take advantage of the relationship to introduce suggestive therapy. We believe that psychological factors are involved in insulin therapy, as in almost every form of medical treatment; however, that the curative effect of such a definitely physical treatment as insulin shock is purely psychological seems *a priori* rather improbable. Furthermore, we question Schatner's explanation, inasmuch as some patients become relaxed and even lucid for short periods after the first small dose, which renders them neither sick nor helpless.

A physiological hypothesis of the effect of metrazol is offered by Meduna and Friedman (**29**). They suggest that the therapeutic effect may be due, in part, to increased cerebral oxygenation as the result of vasodilation. Schilder (**49**) offers a somewhat more psychological interpretation: "The victory over the death threat, expressed in the epileptic fit and lingering on in the perceptual and aphasic difficulty, enables the individual to start life and relations to human beings all over again." He characterizes the therapy by stating: "The treatment is an organic treatment reflected in psychological attitudes."

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# RECENT RESEARCH IN CUTANEOUS SENSITIVITY: I. PAIN AND TEMPERATURE

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Eight years have passed since the literature on cutaneous and kinesthetic sensitivity was reviewed by M. J. Zigler (142) for the *Psychological Bulletin*.[1] In the material which has accumulated since that time, there is apparent an excessive narrowness of approach and a deplorable lack of communication among investigators separated by language barriers or working in different compartments of scientific discipline. This review can serve as a contribution to the progress of cutaneous research if it helps to make known the full scope of the field and the variety of approaches that are available to workers within it. We hope, as well, to crystallize the crucial problems, to provide a critical evaluation of difficulties and contradictions, and to emphasize promising new theories and techniques.

## Pain

In addition to the obstacles encountered in all research on cutaneous sensitivity, the topic of pain suffers from two special difficulties. The first of these is the virtual impossibility of securing stimuli adequate to pain alone. Stimulation which arouses pain typically is adequate for some other sensory process as well. This seems to be the basis for the unsettled nature of many of the problems in the field and makes the standardization and isolation of pain stimuli an urgent task. The second is the affective connotations of the word *pain*; its ancient philosophical usage as the opposite of pleasure still beclouds the issue, even in research presumably dealing with pain as a sensory quality.

*Pain as a Specific Modality.* Another unlaid ghost is the

[1] The sheer bulk of available references necessitates the omission here of kinesthesis (143) and certain other topics which have acquired the status of separate fields of research, as, for example, vibration (44). We may also refer the reader to a number of review articles dealing with various aspects of cutaneous sensitivity; these overlap only in minor topical or chronological respects with ours (1, 2, 8, 9, 98, 112, 114, 116, 119, 120, 135).

view that pain is the result of the stimulation at high intensities of the sense organs of *any* modality [2]—which is still occasionally expressed (**25, 117, 138**) despite the accumulation of negative evidence. During the period under review, for example, it has been shown that the auditory receptors are not involved in 'auditory pain.' Helsmoortel and Nyssen (**61**) produced pain with intense sounds in deaf patients with *cochlear* loss, but could not do so in patients deafened by *middle-ear* defects (see also p. 292, footnote 8).

In regard to specificity, the critical problem today concerns the relation of pain and touch. The possibility that pain is a function of the pressure-sense dates back to the Von Frey-Goldscheider controversy. Von Frey's theory that pain is a specific modality with its own receptors and pathways is opposed to Goldscheider's that pain results from the summation of impulses in tactile fibers. Both protagonists regarded the end-phase of pain adaptation as crucial: Goldscheider held that it consisted of a 'subpainful sensation' *(unterschmerzlicher Empfindung)* or 'pressure' *(Druck)*; while Von Frey found no qualitative change in the experience, but merely a gradual weakening of the intensity of pain until it disappeared.

For this reason, several studies during the period under review have employed pain adaptation to test the opposing theories. Wells and Hoisington (**137**) and Burns and Dallenbach (**21**) both found pressure as a prominent experience in the end-phase of pain produced by needle algometers. However, as Burns and Dallenbach pointed out, crucial results could not be obtained by a technique which provided an adequate stimulus for touch as well as for pain. Stone and Dallenbach (**123**) used radiant heat to produce a mild pain which arose and disappeared gradually—important because Goldscheider had pointed out that sharp, intense pains might mask other sensations. Under these conditions, no experience of pressure was found, the sensory end-phase being merely an experience of warmth. Analogous results were obtained by Edes and Dallenbach (**32**) with 'radiant cold.' The view was expressed that Goldscheider's residual pressures were a function of the stimulus he employed and not a necessary concomitant of pain adaptation.

A major contribution to the analysis of the problem has come from the application of recently developed techniques for the study

[2] Dallenbach has recently systematized the historical background. See (**32**) and Dallenbach, K. M. Pain: history and present status. *Amer. J. Psychol.*, 1939, **52**, 331–347.

of specific fiber groups in peripheral nerve tracts.[3] Directly relevant are the valuable studies by Heinbecker, Bishop, and O'Leary (**54, 55, 56**), whose conclusions are in close agreement with those of Goldscheider. Pain, according to these investigators, is associated with myelinated fibers, three to six microns in diameter, distinct from a fiber group of larger diameters which is related to 'pure' tactile experience. Near-threshold stimulation of the small-fiber group produces a so-called *pricking touch* sensation and ". . . pain ensues when a sufficient number of impulses resulting in a pricking touch sensation are centrally summed." Thus, *two* kinds of tactile experience and nerve fibers are posited, summation in one of these producing pain.

It is our opinion that these findings can be reinterpreted as evidence for the Von Frey theory. Their essential contribution is the definitive dissociation of pain and touch fibers. Heinbecker, Bishop, and O'Leary may have called the small-diameter group 'pricking touch fibers' (instead of 'pain fibers') because of the prevalent view that pain must necessarily be unpleasant or distressing. Thus, threshold *pain* (commonly known to psychologists as *prick, i.e.* mild pain) is designated as a particular kind of *touch*—a word which does not carry the connotation of unpleasantness. Omitting the word 'touch,' which—if we are correct—is unnecessary, leaves us with clear evidence of separate fiber groups for pain and for tactual experience, in line with Von Frey's hypothesis. Rather than a qualitative change to be explained by summation, we may think of a quantitative continuum—at higher intensities becoming, to be sure, unpleasant or even unbearable.

Other evidence likewise points to a dissociation of touch and pain. Raffel (**115**) found complete dissociation in the recovery of sensitivity to touch and to prick following pressure block. Waterston (**134**) was able to slice through nerve fibers and end-organs in the epidermis, apparently subserving touch, without producing pain. Dearborn (**29**) reports a case diagnosed as congenital pure analgesia, in which there was no experience of pain, even from the most extraordinary insults to skin and viscera, although other forms of sensitivity appeared to be normal. Grindley (**45**) found that changing the rate of skin

[3] The techniques and findings of Gasser and Erlanger, of Heinbecker, Bishop, and O'Leary, and of others who have attempted correlations of sensation, fiber size, and characteristics of the nerve impulses will be examined in more detail in another paper, which will deal with touch and the neural bases of cutaneous sensitivity.

deformation affected the threshold for touch but not for pain; yet, according to the summation theory, we should expect the stimulation rate markedly to influence the pain threshold.

*Studies of Pain Using Electrical Stimulation.* Although the following studies involve a variety of research aims, their use of electrical stimulation makes it desirable to discuss them together, because of the common pitfalls. For example, the difficulty of obtaining a stimulus adequate to pain alone, which has been referred to previously, is of obvious importance here. This is evident in such work as that on sensory cutaneous chronaxy, where earlier studies (*e.g.* **133**) confused pain and pressure experiences, either in the determination of the rheobase or in subsequent trials. Neff and Dallenbach (**108**; also **80**), by making the distinction clearly, showed how to obtain chronaxies for pain and pressure that are reliably different.

Another general difficulty arises from the attempt to produce direct stimulation of the nerve fibers, the criterion usually being the streaming or tingling nature of the sensation. Research in this field has not consistently maintained the careful and continuous control through subjective reports demanded by this criterion, nor has it always recognized the possibility (pointed out by Adrian some 20 years ago) that the current may simultaneously excite both nerve fibers and end-organs. We must, therefore, be cautious in accepting the conclusions of Hauck and Neuert (**53**) and of Haimann and Schenk (**48**) in favor of a central summative theory, on the basis of pain produced by multiple electrical stimulations.[4]

Still another complication is suggested by the work of Thompson and his collaborators (**128, 129**), who produced *anesthesia* in the area supplied by a nerve by the unipolar application of alternating current to the nerve trunk through the intact skin.[5] On the other hand, Lewis (as will be detailed in the next section) developed a method of producing *hyperalgesia* by electrical stimulation.

*New Theories of the Neural Mechanisms of Pain and Hyperalgesia.* The proposal by Lewis (**84**) of a new 'nocifensor' system of cutaneous nerves results from an exquisitely conceived series of experiments, the outgrowth of his earlier work on a chemical

[4] Even greater caution is elicited by such elliptical reasoning as the following: Alcohol is found to raise the pain threshold, while hypoglycemia lowers it. Alcohol and insulin are known to produce opposite central effects; therefore, the thresholds investigated must be central thresholds.

[5] Additional evidence for the dissociation of pain and touch is to be found in the report that pain sensitivity disappears at a different current frequency or current intensity than does touch.

theory of hyperalgesia. Basically, Lewis discovered that skin injury or faradization of a cutaneous nerve could produce a long-lasting smarting and hyperalgesia—always localized within, and sometimes completely occupying, the territory of the cutaneous nerve involved. He then set to work painstakingly to eliminate one possible explanation after another: (a) a purely chemical theory (such as that of Lewis and Hess, **87**) could not account for the distribution according to the area innervated by the nerve; (b) the influence of efferent impulses from the central nervous system was excluded by blocking the nerve trunk proximal to the injury, which did not affect the phenomenon; (c) the influence of the sympathetic nervous system was eliminated, because patients with gangliectomies showed no difference on the normal and sympathectomized sides of the body.

However, nerve block distal to the injury did halt the spread of hyperalgesia, showing that local neural connections of some kind were implicated; and an ingenious set of experiments demonstrating the nature of the spread of hyperalgesia with stimulation eccentric to the narcotized region indicated that a branched rather than a network system was responsible. Lewis reaches the conclusion that he has demonstrated the existence of a system of *effector nerves* which he proposes to call, because of their association with local defense against injury, 'nocifensor nerves.' These are believed to be connected with ". . . some such process as the release of stable chemical products at the effector endings of the nerves concerned." In a later article (**85**) he suggests that the demonstration (*e.g.* **6**, **76**) of efferent fibers in the dorsal roots of the cord may provide the anatomical basis for the nocifensor fibers.

It is our opinion that Lewis's experimental results are best explained by a more parsimonious solution, namely: antidromic action in the sensory nerves. Lewis rejected this interpretation on the logical ground that the sensory system for pain is capable of localization and is therefore incompatible with an effect occupying the entire territory of the nerve. However, this matter of localization seems an insufficient basis for hypothesizing an entirely new division of the nervous system, for two reasons: (1) Antidromic action (particularly in response to strong stimuli) may have different characteristics from the usual dromic action of these fibers; and (2) Lewis's own experiments sometimes show a degree of localization of the hyperalgesia, because the phenomenon did not *always* occupy the entire region supplied by the cutaneous nerve.

Nafe and Wagoner (**103**, **106**, **107**) offer an entirely different

suggestion for a new neural mechanism of pain, holding that certain kinds of pain may be produced by a pattern of nerve impulses resulting from extreme vasoconstriction. Their experiments show a correlation between vasoconstriction and pain, but direct evidence that the connection is a causal one is not available. It has long been known that pain is accompanied by vasomotor and other autonomic changes (see, *e.g.* **22**, **109**), although these have generally been taken to be the result rather than the cause of the pain.

*Chemical Factors in Pain and Analgesia.* A considerable number of studies point to the possibility of chemical action in connection with pain. Lewis and Hess (**87**) report ". . . a susceptible state [hyperalgesia] in which pain is easily induced and in which pain occurs spontaneously from time to time," produced by light burning, scratching, or other injuries, with redness, swelling, and tenderness lasting one to six days.[6] Heat accentuates and cold relieves this state, as was also demonstrated by Gammon, Starr, and Bronk (**36**); whereas occlusion of the circulation causes extreme persistence of the pain from mild stimulation. Lewis and Hess postulate the release of a chemical substance into the intercellular spaces which acts to raise the excitability of the pain nerves. Normal circulation acts to 'wash away' this substance; hence the persistence under occlusion. This chemical substance is believed to be different from that causing flare and reddening (**85**), but it has not been positively identified. Lewis's later work leads to the view that it is released by the action of 'nocifensor nerves' (see p. 288). Several writers have suggested that such a chemical factor may be the agent in the production of both local and referred pain and not merely limited to the explanation of hyperalgesia (**28**, **94**, **136**). Rosenthal and Minard (**153**) offer strong evidence for the identification of the substance with histamine, although the absence of histaminase in the skin would make it difficult to account for adaptation.

Charpentier (**26**) speculates, on the basis of research on anesthesia, that analgesia is produced when the central nervous tissue is relatively more dehydrated than peripheral nervous tissue. Jores and Frees (**74**) found a consistent curve of diurnal variation in pain sensitivity (to an electric current applied to the teeth); this is said to parallel the known curve of adrenal secretion. Possibly related is Herren's experiment (**62**), showing a reduced two-point threshold

[6] Undoubtedly the existence of such states accounts for the persistence of the belief that pain is not subject to adaptation. Actually, we have in this condition a prolonged lowering of the pain threshold.

for pressure-pain during the premenstrual period when there is a high concentration of the follicular hormone in the blood stream. Mullin and Luckhardt (**95, 96**) found a marked reduction in pain sensitivity, without any effect on touch sensitivity, following administration of alcohol, morphine sulphate, and codeine sulphate. This rise in pain threshold from the ingestion of alcohol is likewise noted by Haimann and Schenk (**48**), who also report a lowering of the threshold during hypoglycemia. Much of this work must remain of dubious theoretical significance until we know better which effects are specific to the pain sense and which are to be taken as indicators of more general changes in the body chemistry.

*Visceral and Referred Pain.* The main reason for including visceral pain in this review of cutaneous sensitivity is the accumulation of evidence that some referred pains are partly dependent upon cutaneous phenomena.[7] Morley (**94**), Davis and Pollock (**28**), and others report abolition of 'shoulder tip' pain—pain in the superscapular region produced by pressure on the diaphragm—by anesthetizing the *cutaneous region* to which the pain is referred. Although this has been questioned (**141**), the weight of evidence strongly favors the genuineness of the phenomenon. The authors mentioned conclude, independently, that there is a release of some chemical substance (see also p. 290) at the site where pain is felt, stimulating the ordinary sensory endings of the skin. For the present, we may disregard their conflicting opinions as to the path by which the excitation is transferred from the diaphragm to the shoulder tip.

There have been other demonstrations of physiological effects at the locus to which pain is referred. Stürup (**126**) discovered hyperalgesic 'pale zones' on the skin of the chest associated with severe pain produced by dilation of the esophagus. Spiegel and Wohl (**121**) demonstrated an increased electrical skin potential ('viscero-galvanic reflex') in skin areas corresponding to referred abdominal and thoracic pains.

[7] Recent surveys (**5, 19, 94, 126,** etc.) agree that visceral pains arise both in the viscera themselves and by reference to other tissues. Alvarez (**3**) explains the apparent insensitivity of the viscera to cutting and burning by assuming that very few sensory endings are involved; whereas stimulation in the *intact* organism produces pain by reaching ". . . an area wide enough to affect many nerve endings." Moore, working with Moore and with Singleton (**91, 92, 93**), produced a so-called 'pain response' in animals by chemical stimulation of afferent nerve endings in the arteries—a mechanism which is advanced as a possible basis for visceral pain.

*Adaptation of Pain.* During the period under review many studies have confirmed the view that pain is adaptable.[8] Three of these employed needle algometers (**21, 137, 124**), the last a multiple-point algometer. Complete pain adaptation was found in approximately 80% to 100% of the trials. All agree in reporting a tremendously variable adaptation time, both in group data and among the results from a single individual. There is a slight, but unreliable, tendency for adaptation time to increase with heavier loading of the algometer. The 'areal' and 'multiple' pains recorded by Stone and Dallenbach showed much the same characteristics as the punctiform pains of the other experiments.

Thermal pain stimulation was used by Dallenbach and his co-workers (**32, 123**), adaptation again appearing in almost all cases, but with the same great variability in time as evidenced in the needle algometer experiments. With radiant heat and 'radiant cold,' the factors producing variability include not only differential sensitivity in various parts of the skin, but also the spread of stimulation to receptors below the surface and to surrounding regions. This latter was held responsible for the occasional failure of adaptation (by a rigid criterion) in Stone and Dallenbach's study (**123**).

Another aspect of pain adaptation is the effect of prewarming and precooling the skin on the threshold for pain from high temperatures, studied by Ômori (**111**).[9] With large areas, the pain threshold temperature varied with the precooling or prewarming temperature, although not in a linear relationship. With small areas, however, precooling or prewarming had very little effect, and the threshold was lowest at normal skin temperature.

Bykhovskaya and Eidinova (**23**) found a striking influence of the sympathetic nervous system on pain adaptation time. Unilateral blocking of the stellate ganglion or unilateral gangliectomy increased the time for adaptation roughly tenfold in comparison with the unaffected side. This suggests a sensory regulatory function for the autonomic nervous system of the sort advanced by Orbeli,

[8] It is unfortunate that some authors are still unaware of this fact. For example, Helsmoortel and Nyssen (**61**) assumed that pain was not subject to adaptation, and hence concluded that the pain produced by high intensities of sound was caused by reflex tension on the tympanic membrane and its rapid disappearance by relaxation thereof. It is clear that this is by no means a necessary conclusion.

[9] It is unfortunate that Japanese work is not properly represented in any of the reviews in this field; some of it appears in inaccessible journals, and much of it lacks even a summary in an Occidental tongue.

Tournay (**130**), Dusser de Barenne, and Foerster, whose implications need to be thoroughly examined. Jores and Frees's (**74**) suggestion of the influence of adrenal secretion on pain sensitivity may be related.

Several studies of 'long-time' adjustment or adaptation to pain stimulation (**7, 138**) need thorough reworking before their data are worth considering. Failure to distinguish 'pain' from 'discomfort' or 'bearableness' completely confuses the issue.

*Phenomena of Plural Stimulation.* The effect on a pain experience of added stimulation has been studied—rather inadequately as yet—by several investigators. Duncker (**30**) found that a second stimulus (pain, auditory, or kinesthetic) produced a *decrease* in the experienced intensity of the original pain, but not in the intensity of the experienced pressure. He rejects distraction as an explanation in view of the actual *experience* of decreased intensity, but his argument for 'urgency' is somewhat circular. Hauck and Neuert (**53**), on the other hand, found that the pain experience was *increased* with electrical stimulation applied to two fingers, supposedly activating the nerves directly. The discrepancy may be a function of the different types of stimulation or of the difference between end-organ and nerve-trunk stimulation.

Gellhorn and his associates (**42, 43**; see also p. 303) report that errors in pain localization are reduced approximately 35% in the region near 'pinched up' skin; whereas those for touch, cold, and warm localization are increased. Gellhorn explains these results in terms of a dominance principle of spinal irradiation (like Ukhtomski's), so that a hyperactive zone in spinal gray matter 'attracts' impulses from less active regions. Gellhorn's crucial argument is that the results must be due to a *qualitative* change in the pain, since the localization of pain is independent of the strength of the stimulus. However, this is nullified by the fact that Kiesow (**77**) has recently demonstrated that the intensity of the stimulus is a significant factor in pain localization. The relation of Gellhorn's experiments to those of Lewis should be examined. Where Lewis records a hyperalgesia filling the area of the *cutaneous nerve*, Gellhorn (following Goldscheider) finds *spinal* irradiation governing the distribution. These two conclusions seem irreconcilable.

*Localization of Pain.* The controversy regarding the relative accuracy of the localization of pain and of pressure has been re-examined experimentally by Zigler, Moore, and Wilson (**144**) and by Kiesow (**77**). Both used thistle spines, as suggested by Von Frey, to produce 'pure' pain. Both find that pain is better localized than

pressure. Zigler, Moore, and Wilson hold that the pressure-pain complex is better localized than either pain or pressure alone and suggest the theory that the two kinds of impulses are integrated in the cortex. (Kiesow's data are inconclusive on this latter point.)

Particularly in view of this integration theory, the question of the purity of the pain becomes vital. The thistle spines may produce a phenomenologically pure pain or a pressure-pain complex. Hence, pain may seem better localized than pressure, because of the presence of near-threshold pressure (masked?) which improves the localization. Kiesow makes it clear that superior localization of pain is obtained only with strong stimuli, *i.e.* when the likelihood of masked pressure is greatest.

Kiesow notes another difficulty: the spread of pain over the skin from the stimulated point. This he attempts to avoid by securing rapid reports and by casting out trials where such spread appears. It is obvious that a certain danger inheres in this procedure, since it may weight the data in the direction of superior localization for pain by picking out the 'best' trials. The controversy with which both papers are concerned cannot be settled until a pure pain stimulus can be developed, or the pressure sense temporarily eliminated. Lewis (**84**) does this by occluding circulation for 20 to 30 minutes, but his quantitative results are too crude to be decisive.

*Phenomenological Descriptions and Classifications.* Numerous papers provide descriptions of cutaneous pains (**21**, **30**, **32**, 123, **124**, **137**); of visceral pain (**5**, **19**); or of pains in other tissues (**86**, **135**). Many of these experimenters have lost sight of the complicating effects produced by associated *nonpainful* stimuli. It is timely of Lewis and Hess (**87**) to point out anew that any brief pain, however produced, may be felt as a slight *prick,* while prolonged stimulation of the same kind may give a *burning* pain. Only a single cutaneous quality is involved, with variations produced by the time factor and by the size of the area stimulated.

A study in which suggestive hypotheses are coupled with lamentable errors of technique and terminology is that of Wilde (**138**). From the experiences produced by a slowly rising stimulus temperature, Wilde concludes that there are three distinct varieties of pain: *brennen, stichen,* and *kribbeln.* These are presented as definitive, because 8 individuals reported one of the 'pure' forms, while the 11 remaining cases are described as 'mixtures' of the pure forms. Such general agreement seems remarkable, since descriptions of unfamiliar sensations would be expected to produce a still greater

variety of terms. Unfortunately, Wilde fails to state his specific instructions—a critical point of technique in phenomenological studies, but the development of specific descriptive varieties is certainly suggestive. Terminologically, he repeatedly confuses the concepts of pain *(Schmerzhaft)*, unpleasantness *(Unlust)*, and bearableness *(Erträglichkeit)*. He likewise fails to take account of the accessory sensations produced by the kind of stimulus used and of the existence of mild pain (thus, he speaks of '*subpainful* burnings'). His novel neurological theory of pain—postulating a dual system with one part operating according to the Von Frey and the other according to the Goldscheider view—requires much more experimental support than his study provides.

## Temperature

Historically, research in the field of temperature sensitivity has been characterized by many sporadic contributions and few systematic, long-term attacks. Perhaps this is the reason why it is dismissed with a few perfunctory lines in the average elementary text, although it offers unsolved problems which are as complex as those in vision and audition.

*Attempts to Identify Receptors.* The nature of the receptors for thermal sensitivity is still an open question. Histological studies by Bazett, McGlone, Williams, and Lufkin, and by Belonoschkin have given a new lease of life to the periodically revived Von Frey theory that Krause end-bulbs are the receptors for cold and Ruffini cylinders are those for warmth. Bazett, McGlone, Williams, and Lufkin (**12**) found a close correlation between the number of warm and cold spots and the number of Ruffini and Krause endings, and an agreement in the depths of these capsulated endings with the depths calculated for warm and cold receptors from reaction-time measurements. Belonoschkin (**15, 16**) merely showed that there are large numbers of Krause and variant endings in the nipple, which is so sensitive to cold that separate spots cannot be mapped.

Two strenuous objections must be entered to accepting these findings as proof of the Von Frey theory: (1) Neither study shows a correspondence between the *location* of these endings and the location of mapped warm and cold spots. Simple correlation of numbers is merely a quantification of Von Frey's original logic. It will be recalled that, in the one instance where location has been considered (Strughold and Karbe's vital staining experiment), there were some

cold spots which did not correspond to Krause bulbs and some Krause bulbs which did not correspond to cold spots. (2) Of far greater importance is the fact that repeated histological studies have failed to reveal any kind of capsulated endings throughout the skin generally. Even if we accept the Von Frey hypothesis for a few special regions of the body (nipple, conjunctiva, genitalia, etc.), the major problem is still unsolved.

A totally different kind of thermal ending is suggested by Woollard (140). After slicing off five successive thin layers of skin—a total of approximately 0.5 mm.—he discovered a small region which still retained some sensitivity to cold. Beneath was a subepithelial, noncapsulated nerve plexus, which Woollard believes must be the cold receptor. The evidence from this painful operation was necessarily too scanty to justify any final conclusion. Nevertheless, the suggestion that cold sensitivity may *extend* to such a depth is worth further investigation.

Other studies point to a locus for thermal receptors much more superficial than that proposed by Woollard. The experiments of Windisch (139), for example, showed threshold values for warmth much lower with diathermy of dry skin (where greater resistance concentrates the heating at the surface) than when the skin was moistened. Likewise, Hardy and Oppel (50) found that *nonpenetrating* infrared radiation, absorbed within 0.1 mm. of the surface, gives lower threshold values for warmth than the *penetrating* rays. For cold, at least, a superficial location is also indicated by the experiments of Endres (33), who placed a drop of narcotic solution on a mapped warm or cold spot and determined the minimum depth to which the skin had to be punctured with a calibrated needle in order to destroy sensitivity: on the average, 0.07 mm. for cold and 0.56 mm. for warm. It is assumed that the depth of the puncture determines the penetration of the narcotic.

Mathematical computations of receptor depth may also be cited, but these must be accepted with caution because of the speculative assumptions involved. Frohwein (35) and Kaestner (75), from measurements of the time required to reach maximum intensity of experience, confirm Hahn's hypothesis that warm and cold receptors lie at approximately equal depths of less than 0.1 mm. Questionable, however, is the correctness of Hahn's theory that the time to reach maximum experience is a function of receptor depth and the accuracy of a rate of thermal conductivity based on measurements with *dead* skin. (For a survey of Hahn's research and theories, see 47).

Bazett and his co-workers (12) attempted to surmount the latter difficulty by measuring the rate of thermal conductivity with fine thermocouples on each side of the prepuce *in vivo*. While this is a great improvement over the dead-skin measurements, it is not certain that extrapolation of these values yields the true rate for slight depths. Mendelson's measurements of the thermal gradients normally present in the tissues (90) indicate that these are not simple linear functions, but that complex, and sometimes even reverse, gradients may be present. Certainly unjustified is the assumption of Bazett and his associates (12) that receptor depths may be computed by subtracting tactual reaction time from minimum thermal reaction time and dividing by the rate of conductivity. This presupposes that the difference between cold and warm reaction times is solely dependent upon a difference in the depth of the receptors—for which there is not an iota of independent evidence. Nor is it logical to use, as they do, only the *minimum* reaction-time figures for computing receptor depths, and then employ the whole set of reaction times—some of them several seconds in length—to calculate the time required for the receptors to function and the minimum change of temperature necessary to set it off (10).

*Theories of Receptor Action.* Although we have made little progress in identifying the receptors for warm and cold, there has been no lack of theories concerning their mode of operation. Several of these involve some modification of the Ebbecke hypothesis that thermal excitation depends upon a *shift* in the temperature gradient which normally exists from the skin surface inward. Windisch (139) thus explains the relative ineffectiveness of wet-skin diathermy, as compared with dry, on the ground that in the former case the skin is heated homogeneously; thus, the temperature as a whole is raised, but the gradient slope is unaltered. However, the critical time relations have not been determined; it may be simply that wet diathermy raises the temperature at the level of the receptors too *slowly* to cause excitation.

Hardy and Oppel (51) give a somewhat similar explanation of their results with radiant energy. Radiometric measurements show that the threshold amounts (for warmth) of three kinds of radiant energy—visible light, penetrating infrared, and nonpenetrating infrared—cause different changes in surface temperature. According to their assumptions, however, each should cause about the same shift in the slope of the temperature gradient, thus accounting for stimulation. These critical shifts in gradient slope have not been measured

*directly,* and again the results might be more simply explained in terms of thermal changes at the receptors. In one article (**49**) Hardy and Oppel suggest that the thermal receptors are differential end-organs, one sensitive spot in a vascular bed at 0.1 mm. and the other at 0.3 mm.—the blood vessels acting like radiators on a thermopile to make the receptors effective over more than their actual area. A curiously similar speculative hypothesis is proposed by Bourgignon (**20**) to explain certain complex results obtained in chronaxy determinations. He holds that temperature sensitivity involves a shift in the temperature gradient between free nerve endings as the upper elements and Meissner corpuscles as the lower. This seems to be the first time in history that the Meissner corpuscles (usually assigned to touch) have become involved in temperature sensitivity.

A wholesome note of caution is injected by Bazett and McGlone (**11**), who point out that any simple shift-of-temperature-gradient theory cannot be entirely satisfactory. Intense warmth, they showed, can be experienced upon the release of asphyxial blood following stasis, with the arm immersed in a bath at blood temperature so that no temperature changes can occur. They suggest that some chemical factor in the asphyxial blood must be responsible in this case, and that a similar chemical factor may be an intermediary in normal excitation.

In view of the chemical theories of pain (see pp. 290–291), it is interesting to note that there have been several attempts to picture the operation of thermal receptors in terms of chemical activity. Thus, Kleinsteuber (**78**) presents a physicochemical interpretation of Hahn's theory which seems to be intended as an analogy rather than as a description of the actual processes. O'Connor (**110**), arguing from the variations in cold sensitivity which follow different degrees of prewarming and precooling the skin, suggests that the cold-receptor operation involves three phases of chemical activity. Unfortunately, the three maxima of sensitivity on which he bases this hypothesis appear to depend upon an arbitrary grouping of the data. To an impartial eye, his highly variable figures form a broad band with only one unequivocal mode. Jenkins (**63, 66**) postulates mechanisms for cold and for warm which involve pseudoreversible reactions. The recovery phase requires the presence of an additional substance. This theory grows out of research on adaptation and has proved useful in suggesting research attacks, although some findings are not readily explainable in its terms.

A relation between pain sensitivity and autonomic control has previously been noted (see p. 292). Krogh (**81**) suggests a similar relation between cold sensitivity and the sympathetic nervous system. He interprets the 'reflex cold' which follows stimulation of the pharynx as evidence that the sensitivity of the cold receptors can be suddenly increased by sympathetic impulses, because his thermocouple measurements reveal no concomitant changes in skin temperature as a result of vasoconstriction. Unfortunately, thermocouples are not adequate to establish the exact time relations nor the thermal changes at the level of the receptors. However, the possible direct action of the autonomic nervous system appears to be a fruitful avenue for further research.

Nafe's vascular theory of thermal sensitivity (**100**) takes the drastic step of eliminating all specific thermal receptors, making warmth simply the kinesthetic pattern from dilating blood vessels and cold, from vasoconstriction. The principal support for this hypothesis comes from a series of experiments by Nafe and his colleagues showing that temperature sensitivity and blood vessels are both absent from the cornea (**101, 103**), that there is a close correlation between thermal sensitivity and vascular activity (**102, 131, 132**), and that the failure of one is linked with the failure of the other (**104, 105**). The theory has been attacked by Jenkins (**72**) on the ground that Nafe's research does not conclusively establish a causal relationship between vascular activity and temperature experience, and that there are a number of experimental facts which cannot readily be explained in terms of vascular action.[10] Nafe has replied (**99**), point for point, to his criticisms, maintaining that a causal relation is the best assumption from the evidence, and that the suggested negative points can all be explained in vascular terms. Further evaluation of the theory seems to wait upon the accumulation of a greater body of critical evidence.

*Adaptation.* Continued research increasingly reveals the complexity of phenomena of thermal adaptation, supporting the concept of a complex underlying process. Although adaptation in large areas is typically slow and incomplete, Dallenbach and his co-workers (**4, 83**) discovered that individual warm and cold spots adapt rapidly and completely with either continuous or intermittent stimulation. Jenkins (**63**), using a neutral field surrounding the stimulator to

[10] For example: paradoxical warm and cold; chemical, mechanical, and electrical stimulation; complex adaptation phenomena; the brevity of the critical period for warm 'flicker'; and the shortness of the reaction time to cold.

prevent spread, found that continuous or intermittent cold stimulation with a small stimulator caused complete adaptation or prolonged depression of sensitivity. Temporary restoration could be effected by brief application of a high temperature. Gradually lowering the surrounding field temperature caused the spot to become suddenly insensitive at a critical level, while raising the field temperature sometimes heightened, but more often depressed, sensitivity.

In a series of experiments with circular, rectangular, and annular stimulators (64, 65, 66, 67), Jenkins showed that the time required for complete adaptation to cold and warm was a function not only of the area but also of the shape of the stimulator. In some cases, adaptation time was proportional to the perimeter of the stimulator; in others, to the square root of the area; and examples of combined effects could be found. It becomes evident from these complicated relationships that the simple classical theories of adaptation are inadequate. Neither the Weber theory (attainment of physical temperature equilibrium) nor the Hering theory (shift of the physiological zero) provides a satisfactory explanation. It seems probable that an adequate theory of adaptation can be written only in terms of the probable receptor processes involved.

*Mapping.* Supplementing earlier data for cold, Earhart and Dallenbach (31) demonstrated that an interval of 20 minutes between successive warm-spot mappings was adequate to eliminate artifacts produced by adaptation. Strughold and Porz (125) made an exhaustive study of the average number of cold spots on 58 regions of the body, finding an extreme variation of 0.7 to 19 per cm.$^2$, with a mean of 7. Münch and Schriever (97) confirmed the view that the thermal sensitivity, sometimes incorrectly attributed to the teeth, is actually that of the gums, the teeth themselves yielding only pain in response to extreme temperatures.

With a mechanical mapping device which permitted accurate control of temperature, pressure, and duration, Heiser showed that the number of mapped warm spots on the forehead increased with higher temperatures of stimulation (57), but was only slightly affected by changes in stimulus pressure (60) and in stimulus duration (58). As the temperature was raised, the new spots which appeared were usually adjacent to those responding at lower levels. At the highest temperatures, warmth or heat was reported from virtually the entire area in some cases. Heiser points out that, while his results do not disprove the punctiform theory of temperature sensitivity, they are congruent with a nonpunctiform hypothesis.

Jenkins (**147**, **148**) has developed a method of *seriatim* mapping with stimulators of different sizes. A checkerboard pattern is mapped repeatedly, and the sum of the reports (0, 1, 2, or 3 intensity) for each square treated as a score. The repetition permits checking the consistency of reporting, and preliminary tests with untrained individuals have shown a high reliability. Maps plotted from the scores show hills and valleys of sensitivity, rather than isolated warm and cold spots. Jenkins suggests discarding the traditional *punctiform* theory (*i.e.* 13–15 cold spots and 1–2 warm spots per cm.$^2$, each supposedly corresponding to an individual receptor) in favor of a *concentration* theory (*i.e.* hundreds of minute receptors per cm.$^2$, with the intensity of experience depending upon their concentration in the area stimulated).

*Paradoxical Warm.* Although the phenomenon of paradoxical cold is generally accepted, the existence of paradoxical warm has often been questioned. Grundig (**46**) failed to obtain paradoxical warm under 'favorable circumstances'—that is, during the stages of local anesthesia after cold sensitivity had disappeared. Although some warm sensitivity was still demonstrably present, it was probably already in a depressed state, so the negative results are not surprising. On the positive side, Pavlicek and Jenkins (**113**) stimulated mapped warm spots alternately with 26° and 39° C. and obtained similar reports of warm from both temperatures in 29 out of 33 naïve individuals. As a by-product of seriatim stimulation at 18° C., Jenkins (**71**, p. 420) cites 10 cases where reports of paradoxical warm occurred consistently from a single square of a checkerboard map. A crucial test would require interspersed contacts with a stimulator at skin-neutral, as a control against suggestion. However, these results, when coupled with the earlier positive findings of Rubin and of Goldscheider, seem to confirm the reality of the phenomenon of paradoxical warm.

*Heat.* New measurements of the liminal temperatures for the experience of heat, made by Lowenstein and Dallenbach (**88**), disclosed one individual with a limen only slightly above 40° C. It is also generally recognized that reports of 'heat' can be elicited from simultaneous stimulation with mild warm and mild cold, and improved demonstration grills for this purpose have been described by Freeman (**34**) and Koch (**79**). Naïve individuals, however, do not always report 'heat' in response to such compound stimulation. Jenkins (**68**, **69**, **70**) obtained negative results except in a scattered minority of cases.

According to the Alrutz theory, the unique experience of 'heat' depends physiologically upon simultaneous excitation of warm and cold receptors, the latter paradoxically stimulated, and cannot be experienced where no cold receptors are present. Jenkins (71) attempted a crucial check of this theory by seriatim stimulation. Naïve individuals consistently reported 'hot' or 'heat' from squares of the checkerboard which were consistently 'neutral' to low temperatures. In the only available study with *trained* observers, moreover, similar results were secured. Heiser (59) found that two trained observers reported 'heat' from spots which showed no cold sensitivity within 2 mm.

Such data suggest the inadequacy of the Alrutz theory, but there remains the new task of accounting in some other way for the reports of 'heat' so consistently obtained in the earlier warm-cold grill experiments. Tentatively, it is suggested that this 'synthetic heat' may not be identical with the normal heat experienced from high temperatures, but is sufficiently similar for the two to be confused. In any event, the nature of an experience of such high intensity, obtained from mild warm and mild cold, is worth further investigation in its own right.

*Threshold Measurements.* Following earlier and less precise work by Bohnenkamp and Pasquay (17), direct radiometric measurements of the radiant energy thresholds for warmth have been developed by Hardy and Oppel (52). Up to approximately 200 cm.$^2$ they find a reciprocal, but not linear, relation between energy and area, indicative of a partial summation effect, which holds in some cases with nonadjacent skin areas such as the two hands. At 200 cm.$^2$ a certain minimum energy is required per unit area, which is not reduced by employing larger areas. This minimum they identify with the limen for the most sensitive receptors. In a series of briefly reported studies with intermittent radiant warm stimulation, Geblewicz (37, 38, 39, 40, 41) discovered a reciprocal relation between duration and logarithm of intensity which is approximately linear (as in visual flicker) when adaptation artifacts are excluded. He also showed that under optimum conditions the critical period for warmth-flicker could be as low as 0.15 second. Belonoschkin (14) found that solutions of magnesium chloride, subcutaneously injected, reduced sensitivity to both cold and warm—a specific effect of the drug and not an artifact of injection.

*Genetic Studies.* Tests of temperature sensitivity in young human infants have been reported by Jensen (73) and by Crudden (27).

The latter is particularly noteworthy, because tactual concomitants were excluded by changing the temperature in a capsule attached to the leg, and the responses were recorded both by protocol and by motion pictures. Although quantitative interpretation was not attempted, rapid adaptation to both warm and cold was demonstrated. Interesting as the first systematic study of the development of temperature sensitivity during the fetal period is the research of Carmichael and Lehner (**24**), who found a sharp increase in the responses to thermal stimulation during the mid-period of fetal growth in the guinea pig.

*Phenomena of Plural Stimulation.* Goldscheider discovered some years ago that, with a warm or cold stimulator held against the skin, temperature experiences could be elicited from the surrounding region by simple tactile stimulation. Following this suggestion, Taeger (**127**) mapped the boundaries of the area around a primary thermal stimulator within which such effects could be obtained. His results show an almost suspiciously regular ovoid field, which is independent of the region of the body used and the intensity of the primary stimulus, but which varies with the area of this stimulus and differs somewhat for warm and cold. To what extent the influence of suggestion was avoided is not made clear in his report. The inhibitory effect of pain was demonstrated by Gellhorn and Northup (**43**), mildly painful stimulation raising the threshold for both warm and cold (see p. 293).

McFadden (**89**) suggests a relation between temperature sensitivity and gustation, because the gustatory reaction time to various concentrations of NaCl was shorter when the solutions were warm—an effect that might depend merely upon physical absorption. Bellows demonstrated (**13**) that the critical frequency for tactual flicker was affected by the temperature of the air jet. Minimal values were obtained with temperatures close to skin neutral, which suggests simple distraction rather than a specific inhibitory influence of temperature sensitivity.

*Perceptual Phenomena.* Bohnenkamp and Schroer (**18**), in a comparison of fixed and moving radiant stimuli, found that movement in itself did not lower the threshold for warmth, as had been suggested. Schroer (**118**) showed that a 9-mm. stimulator could be consistently discriminated from a 13-mm. stimulator when applied either to corresponding or noncorresponding parts of the body. Stone (**122**), on the other hand, demonstrated that the perception of thermal *form* as such is very poor. With both radiant and contact

stimulation, the reports showed only slightly better than chance agreement with the actual shape of the stimulated area. Lauterbach and Crouser (82) disproved the traditional statement that 'wet is a blend of cold and pressure,' by showing that 'wet' could be consistently reported in the absence of any thermal component whatsoever.

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# ON ELECTION TO MEMBERSHIP IN THE AMERICAN PSYCHOLOGICAL ASSOCIATION

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At the Stanford meeting of the American Psychological Association, Inc., in September, 1939, it was voted "to create a Committee on the Constitution to reconsider the by-laws pertaining to the qualifications and privileges of Associates and Members . . ."[1] One reason for the creation of this Committee is the fact that the numbers of Members and Associates seem to be so badly out of balance. The 1939 *Yearbook* of the Association—the last available at the moment of writing—indicates that there were 618 Members and 1909 Associates in 1939, and at the 1939 meeting 53 Members (most of whom were previously Associates) and 257 new Associates were elected. Hence, the 1:3 ratio between Members and Associates seems likely to be maintained under the present conditions and even may tend to increase.

Actually, the difference between the rights and privileges of Associates and Members is threefold: Members only may (1) hold office, (2) exercise the franchise, and (3) participate in the assets of the Corporation (although this point has never been raised to my knowledge). Associates have all of the other privileges of Members, namely: the right to attend and read at scientific meetings and the right to receive the same printed matter. The by-laws of the Association provide that Members must have the Doctor of Philosophy degree "based in part upon a psychological dissertation" and also present "acceptable published research of a psychological character beyond the doctorate dissertation."[2] This latter qualification has never been rigidly defined, but it has been rather strictly and severely interpreted by succeeding Councils of Directors to mean at least two major psychological publications beyond the doctorate dissertation. The Council of Directors considers the case of every candidate and recommends to the business meeting of the Association those candi-

[1] Cf. *Psychol. Bull.*, 1939, **36**, 753 f.

[2] Cf. *Yearbook* of the American Psychological Association, 1939. P. 21, Article I.

dates whom it believes qualified for Membership or Associateship. These by-laws make the standards for election to Membership so high that anyone elected a Member of the American Psychological Association is considered to be qualified as a Fellow in the American Association for the Advancement of Science.

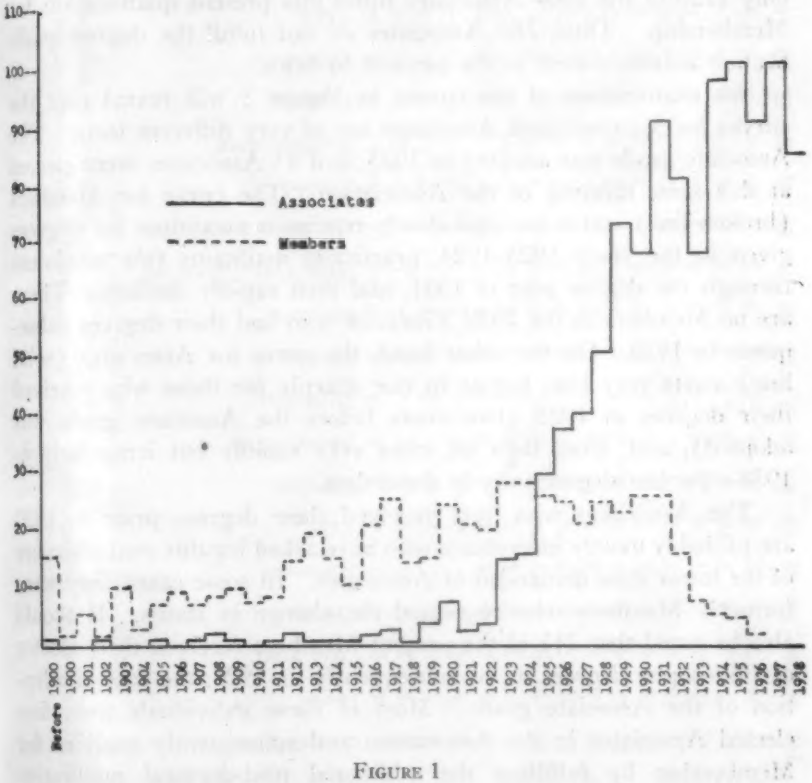


FIGURE 1

DISTRIBUTION OF MEMBERS AND ASSOCIATES OF THE AMERICAN PSYCHOLOGICAL ASSOCIATION, INC., WITH AMERICAN DEGREES OF DOCTOR OF PHILOSOPHY IN TERMS OF THE YEAR IN WHICH THE DEGREE WAS GRANTED

It would seem that it is the post-doctoral publication qualification which has prevented many Associates from becoming Members of the Association. In Figure 1 will be found the distribution of the dates of receiving the Doctor of Philosophy degree for both Members and Associates. Two Members who do not give the dates of their Ph.D. degrees are not included and, in the curve for Associates, only those with the Ph.D. are included. The curves are for American institu-

tions only. This excludes 25 Associates who have the degree of Doctor of Education, in one of its several forms, together with 37 Associates and 36 Members with degrees from foreign Universities. The data were obtained from an analysis of the individual reports in the 1939 *Yearbook* of the Association. The results show that 610 of the 618 Members hold the degree of Doctor of Philosophy, while only 1120 of the 1909 Associates fulfill this present qualification for Membership. Thus, 789 Associates do not fulfill the degree qualification as established in the present by-laws.

An examination of the curves in Figure 1 will reveal that the curves for Members and Associates are of very different form. The Associate grade was adopted in 1925, and 45 Associates were elected at this same meeting of the Association. The curve for Members (broken line) starts low and slowly reaches a maximum for degrees given in the years 1923–1924, practically maintains this maximum through the degree year of 1931, and then rapidly declines. There are no Members in the 1939 *Yearbook* who had their degrees subsequent to 1936. On the other hand, the curve for Associates (solid line) starts very low, begins to rise sharply for those who received their degrees in 1923 (two years before the Associate grade was adopted), and, from then on, rises very rapidly but irregularly to 1938—the last degree year in these data.

The Associates who have received their degrees prior to 1920 are probably mostly individuals who have asked for this status because of the lower dues demanded of Associates. In some cases, they were formerly Members who requested the change in status. It should also be noted that 211 of the present Members received their Doctor of Philosophy degrees in, or subsequently to, 1925, the date of adoption of the Associate grade. Most of these individuals were first elected Associates in the Association and subsequently qualified for Membership by fulfilling the additional post-doctoral publication requirement. But the rapid drop of the Members' curve for the 1932 degree date and for subsequent years indicates that there is a serious lag of five to six years after the granting of the degree before such qualification is usually possible.

It has seemed worth while to analyze the Associate list with regard to both the dates of the Ph.D. degrees and the institutions which granted them. These data will be found in Table I. There are only 69 Associates who obtained this degree prior to 1925, and these are entered as a group in the first row of the table. In the first 20

TABLE I

DISTRIBUTION OF ASSOCIATES OF THE AMERICAN PSYCHOLOGICAL ASSOCIATION HOLDING THE DEGREE OF DOCTOR OF PHILOSOPHY FROM AN AMERICAN INSTITUTION IN TERMS OF THE DATE OF THE GRANTING OF THE DEGREE AND OF THE INSTITUTION GRANTING THE DEGREE

| | Columbia | Iowa | Ohio State | Chicago | Minnesota | Yale | Harvard | Pennsylvania | Hopkins | Michigan | Stanford | New York University | California | Peabody | Northwestern | Cornell | Wisconsin | Clark | North Carolina | Princeton | 36 Other Institutions | Total by Years |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Before 1925 | 9 | 4 | 1 | 6 | 1 | 4 | 3 | 4 | 7 | 1 | 2 | — | 4 | 1 | — | 5 | 1 | 3 | — | 1 | 12 | 69 |
| 1925 | 7 | 1 | 2 | 3 | — | 1 | — | 2 | — | 3 | — | 1 | — | 1 | 2 | 1 | — | — | — | — | 6 | 30 |
| 1926 | 8 | 5 | 1 | 6 | — | 1 | 4 | — | 2 | 1 | 1 | — | 1 | 1 | — | 1 | — | 1 | 1 | 1 | 2 | 37 |
| 1927 | 8 | 1 | 4 | 5 | — | 2 | — | 5 | — | 2 | 1 | 2 | 1 | 3 | 1 | — | — | — | — | — | 5 | 40 |
| 1928 | 6 | 6 | 7 | 9 | 4 | 1 | 2 | 3 | 2 | 1 | 1 | 3 | — | — | — | 1 | 2 | — | — | — | 4 | 52 |
| 1929 | 14 | 7 | 5 | 8 | 4 | 3 | 2 | 4 | 3 | 3 | 3 | 3 | 1 | 1 | — | 2 | 1 | 1 | — | 1 | 7 | 73 |
| 1930 | 15 | 1 | 9 | 5 | 5 | — | 4 | 3 | 2 | 3 | 3 | — | 2 | 5 | 1 | — | 2 | — | 3 | — | 3 | 66 |
| 1931 | 15 | 6 | 17 | 6 | 3 | 4 | 3 | 1 | 3 | 3 | 3 | 3 | 3 | 2 | 1 | 1 | 1 | — | 2 | — | 13 | 90 |
| 1932 | 16 | 12 | 5 | 1 | 5 | 1 | 3 | 5 | 4 | 2 | 2 | 2 | — | — | 4 | 1 | — | — | — | 3 | 15 | 81 |
| 1933 | 12 | 9 | 4 | 5 | 3 | 6 | 4 | 2 | 1 | 1 | 1 | — | 2 | 2 | 2 | 2 | — | 1 | 2 | 1 | 8 | 68 |
| 1934 | 18 | 5 | 6 | 4 | 4 | 5 | 9 | 1 | 2 | 3 | 5 | 1 | 1 | 4 | 3 | — | 2 | 2 | — | — | 23 | 98 |
| 1935 | 25 | 11 | 8 | 2 | 7 | 7 | 5 | — | — | 1 | 2 | 4 | 1 | 2 | 2 | 1 | — | 5 | — | 2 | 16 | 101 |
| 1936 | 16 | 11 | 6 | 5 | 5 | 4 | 3 | 2 | 5 | 2 | 2 | 5 | 3 | 4 | — | 2 | 5 | 1 | — | 2 | 8 | 91 |
| 1937 | 12 | 8 | 7 | 3 | 9 | 10 | 6 | 2 | — | 4 | 3 | 4 | 5 | — | 6 | 1 | 1 | — | 1 | — | 19 | 101 |
| 1938 | 11 | 9 | 2 | 5 | 8 | 4 | 1 | — | 2 | 2 | 3 | 3 | 3 | — | — | 1 | 2 | — | 5 | 2 | 23 | 86 |
| Total by Institutions | 192 | 96 | 84 | 73 | 58 | 53 | 49 | 34 | 33 | 32 | 32 | 31 | 27 | 26 | 22 | 19 | 17 | 14 | 14 | 13 | 164 | 1083 |

columns are given the data for the 20 institutions which have granted the largest number of Ph.D. degrees to Associates. In the next column are grouped the Associates who received the Ph.D. degree from 35 other institutions in America. These institutions, with the number of degrees given by each to the present list of Associates, are given below:

| | |
|---|---|
| 12 | Illinois |
| 11 | Indiana |
| 10 | Nebraska |
| 8 | Catholic, Pittsburgh |
| 7 | Kansas, Southern California |
| 6 | Bryn Mawr, Duke, Fordham, Kentucky, Virginia |
| 5 | Brown, George Washington, Pennsylvania State, Radcliffe, Rochester, Texas |
| 4 | Boston |
| 3 | Washington, Western Reserve |
| 2 | Cincinnati, Colorado, Hartford Seminary, Missouri, North Dakota, Smith, Vanderbilt |
| 1 | Claremont, Iowa State, Loyola, Washington (Mo.), Notre Dame, Oregon, St. Vincent |

There are marked differences in the number of degrees assigned to different institutions. For example, Columbia, with 192 degrees, has just twice as many as Iowa, which is in second place. Seven institutions (Columbia, Iowa, Ohio State, Chicago, Minnesota, Yale, and Harvard) account for 605, or almost 56%, of the American Ph.D. Associates. And 12 institutions (the seven above, plus Pennsylvania, Hopkins, Michigan, Stanford, and New York University) account for 767, or almost 71%, of the Associates who received their degrees from American institutions.

Comparing the rank order for the volume of degrees in this table with a similar analysis made 11 years before for Members and Associates in the Association from the 1928 *Yearbook*,[3] certain changes of position of institutions are evident. For the present data, Cornell and Clark drop out of the high 12 institutions (from 4th and 5th positions to 14th and 16th, respectively), while Minnesota and New York University in 1939 go up into the high 12 (from 14th and 24th to 5th and 12th positions). Columbia is found at the top of both lists, and the position of Pennsylvania (8th) remains unchanged. Those

[3] Fernberger, S. W. Statistical analyses of the Members and Associates of the American Psychological Association, Inc., in 1928. *Psychol. Rev.*, 1928, 35, 447–465.

institutions losing in rank between the 1928 and 1939 (Associates only) lists are Chicago, Clark, Cornell, Harvard, Hopkins, and Stanford. Those institutions in a position higher in 1939 than they were in the 1928 list are Iowa, Michigan, Minnesota, Ohio State, New York University, and Yale. In general it would seem that the Midwestern institutions have built up volume of degrees at the expense of the Eastern institutions.

It would seem that these results may be of great interest in establishing bases for changes in criteria for election to Membership in the American Psychological Association. One would hardly question the value of the degree of Doctor of Philosophy when granted by any one of the 55 American institutions which contribute the data for Table I nor of that granted by the 14 foreign institutions who have given degrees to the 37 additional Associates with the Doctor of Philosophy degree whose results are not listed in either the table or the figure. It is true that many of these Associates would not care to become Members and thus assume the additional annual dues that such election would entail. How many such there are could be determined only by circularizing the present Associates. For the sake of argument, let us assume that every Associate would accept Membership if it were offered to him.

If the publication criterion should be removed and if the grade of Membership should be offered to qualified Doctors of Philosophy in Psychology, several degrees of restriction might still be applied. Thus, presumably, it might be reasonable to ask such an individual to remain an Associate for a specified number of years before he was elected a Member, unless possibly he could qualify before that time on the basis of some publication criterion. In this case, the results would be as indicated below on the basis of the present Associate group including those individuals with the Doctor of Education degree as well.

| | Members | Associates | Now Qualified as Members Under the Present Plan |
|---|---|---|---|
| All Ph.D.'s as Members..... | 1759 | 768 | — |
| Ph.D. plus 5 years..... | 1258 | 1269 | 13 |
| Ph.D. plus 10 years..... | 864 | 1663 | 102 |
| Ph.D. plus 15 years..... | 675 | 1852 | 227 |
| As at present..... | 618 | 1909 | — |

Clearly, these results seem to indicate that the dropping of the publications criterion for election to Membership and, at the same

time, adopting the substitution of a "period of apprenticeship" as a criterion for such election would not considerably change the present unbalanced condition between the two grades unless the apprenticeship period was made of relatively short duration. And even if the Associate had to wait only five years after his Ph.D. before he was elected to Membership, the present situation indicates that the numbers in these two grades would only be about equally balanced.

The writer has no suggestions to make, particularly in advance of the report of the Association's Committee charged with studying this and other matters connected with the by-laws. He merely presents the factual data so that the problem may be approached objectively.

# BOOK REVIEWS

ROETHLISBERGER, F. J., & DICKSON, W. J., with the collaboration of H. A. Wright. Management and the worker: an account of a research program conducted by the Western Electric Company, Hawthorne Works, Chicago. Cambridge, Mass.: Harvard Univ. Press, 1939. Pp. xxiv+615.

Only rarely does a book appear which transcends significantly the limits of the field in which it was written and illuminates fields ordinarily regarded as foreign to it. *Management and the worker* is such a volume. Although its title would tend to attract the interest only of the industrial psychologist, the fact is that the social psychologist, the clinical psychologist, the economist, and the sociologist will be richly repaid for the time they spend in studying its 600 pages of original material.

This is no general treatise on management. It is not a rehash of classical economics and atomistic industrial psychology. It is an honest, clearheaded, and precise account of a series of detailed, firsthand investigations into the social psychology of an industrial plant extending over a decade. True, some of the material has already been reported by Mayo and by Whitehead in volumes of recognized importance. In the present report, however, all the materials made available by years of integrated researches are brought together for the first time and viewed as a meaningful whole. And the final result does not fall at all short of what one might have expected from a group of capable, serious-minded investigators who spent years at the task.

It is impossible to describe even the major patterns of the research within the limits of a brief review. There are few of the 600 closely written pages that will fail to excite marginal notations on the part of the careful reader. This is no book to be skimmed lightly in the course of an evening. It must be read slowly, and the reader will inevitably find himself making his own index to supplement the good index provided by the authors. If this reviewer is any criterion, the hours spent in reading will be regarded as well spent when the reader finally begins to sense the import of the analysis and to see how the general thesis permeates many fields other than that of industrial production. If, by way of trivial example, the reading does not provide a better insight into relationships within a university faculty, the reader may feel sure that he has missed some major points.

The starting point for the investigation is familiar enough to warrant only general mention, for both Mayo and Whitehead have discussed it at some length. The Harvard group was set at a 'routine' problem—that of investigating the effects of rest-pauses and varied lengths of working day on a group of workers. Their initial proposals would have won approval from any of those who still feel that the psychologist may ape the physical scientist and think in terms of single variables. A group of girls was isolated and the study explained to them. Early results were

as expected: rest-pauses were accompanied by increased output. Then the concept of easy investigation collapsed, for a return to the original conditions, which should have brought about reduction of output, witnessed, instead, a continued increase. This pointed to variables not included in the original plan, and Mayo has described well the steps that were taken to study the social identification of the worker with his work.

This is really the starting place of this volume. The facts of the relay assembly room are reviewed and discussed. Then the reader is taken through the successive steps which, over a period of years, were intended to investigate the social organization represented by the workers at their work. It is a frank account which depicts the shortcomings of the several stages and indicates how slowly the final hypothesis began to force itself into the thinking of the researchers. Thus the reader follows through the studies in a second relay assembly room, through a program of interviewing which finally embraced detailed interviews with 21,000 employees, through investigations in the mica-splitting room, and most important of all, through a long term of carefully planned observations in the bank-wiring room. In each of these units the development of the method of investigation is clearly portrayed, and the results are discussed in terms of hypotheses which become more and more explicit as the results come in. If the idle reader wishes a sample of this method of exposition, he may find a good choice in Chapter XIII, which discusses the interviewing technique in a fashion that has wide implications for other fields.

It is peculiarly dangerous to extract from context single items that stand originally in 600 pages of detail, but the risk must be run in order to indicate the major trend of the exposition. There is no better way than to attempt to sketch the major outline by a series of quotations:

"The point of view which gradually emerged from these studies is one from which an industrial organization is regarded as a social system." "Just as each employee has a particular physical location, so he has a particular social place in the total social organization." "As patterns of behavior become crystallized, every object in the environment tends to take on a particular social significance." "According to this way of looking at things, material goods, physical events, wages, hours of work, etc., cannot be treated as things in themselves. Instead they have to be interpreted as carriers of social value." "The function of management, stated in its most general terms, can be described as that of maintaining the social system of the industrial plant in a state of equilibrium such that the purposes of the enterprise are realized." "Overt or verbal behavior at work was no longer regarded as the effect of some simple cause (fatigue, monotony, or supervision) but as the resultant of the interaction of a number of variables making for or against equilibrium."

Gradually and deliberately the reader is made to grasp the importance of the social equilibrium that develops within the informal social organizations in the plant. The resulting social hierarchy is studied in detail. It is shown that the introduction of any change, even one that is intended to be favorable to the worker, may disturb this equilibrium with detrimental effects unless its mode of introduction and rate of introduction are controlled in terms of maintaining this equilibrium. An intimate study of restriction of output in the bank-wiring room makes it apparent that

this restriction is aimed at maintenance of equilibrium within the informal social group and that it is not the result of laziness or of an animus of labor against capital. The various levels of supervision are scrutinized in the light of this dynamic interpretation with profitable results. It is shown that both the transmission of orders downward and the projection of information upward are distorted by the forces of social equilibrium. In the contrast between interpretations in the light of this theory and those in accord with the theories of formal economics, the latter come off rather badly. Yet all of this is presented, not with the haste and fervor of the evangelist, but is soberly advanced as a series of frames of reference inexorably forced upon the investigators by the demands of the data themselves. The outcome is an integrated view of the influences at work in industrial life which would seem to have quite as much significance for the economist, the social psychologist, or the sociologist as for the student of industrial psychology.

The volume in its entirety commands lyric praise from one who has been accused of writing only vitriolic reviews. It is coherent, consistent, and frank, avoiding the dangers of evangelism on the one hand and the pitfalls of dull pedantry on the other. The method of approach employed permits the reader to follow chronologically the thinking of the investigators and to retrace with them the steps by which a given position was reached. The outstanding fault of the volume—and it is a fault shared with another recent publication from the same general source—is a failure to coördinate the findings of these researches with those of other investigators. A reader from Mars would have the impression that the various topics were here being studied for the first time. Granted that other studies have often been less thorough, granted that few of them have approached a dynamic point of view, nevertheless, other investigations of importance do exist. Restriction of output, the use of detailed interviews, the effects of wage-payment plans, the social organization within the group—the treatment of these and many other topics would have gained if the result reported here had been specifically aligned with the existing literature. As it is, the documentation of the volume may be best described as incidental.

Grant these faults and any others that critical eyes may detect and the volume will still stand out as a landmark of real importance in the development of a dynamic social psychology. It will be much to be regretted if the title tends to limit readership to those within the confines of industrial psychology. There are few psychologists, whatever their interests, who could fail to be stimulated and broadened by prolonged and serious contact with the facts and theories contained in its pages.

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CREEDY, F. Human nature writ large: a social psychologic survey and Western anthropology (with a Foreword by B. Malinowski). Chapel Hill: Univ. North Carolina Press, 1939. Pp. ii+484.

Here is a book which a leading anthropologist, Malinowski, terms "one of the most important recent contributions to social psychology."

But social psychologists may well consider it, rather, a treatise on anthropology, social philosophy, or even theoretical economics or religion. The author himself expects it to serve as "a liberal education for those who must live in that [Western] culture." In his "super-optimistic moments" Creedy has wondered whether his treatment of Western culture might not be more satisfactory for university work than that found in the usual liberal arts courses. The reviewer must admit that his optimism does not climb to this extent, although he feels that the intelligent university senior can profit quite considerably from a perusal of this book, particularly *after* he has had many contacts with work in the liberal arts.

Creedy's central thesis is that human happiness is a function solely of physical well-being plus an absence of conflicts. The latter demands the satisfaction of man's basic urges, which are assumed to be the self-regarding, the social, the constructive, and the sexual. These urges alone constitute absolute values. All else is relative. Thus, the principles often assumed to be basic in the fields of religion, politics, patriotism, polite society, business, and the family are termed mythologies and are analyzed into "trigger phrases," linguistic stimuli which set off socially approved (that is, expected) responses. By means of these stimuli we are trained to accept almost without question our social roles.

Six chapters are devoted to "The Struggle of Creative Imagination Against Habit." It is assumed that heavy physical toil is the most complete preventative of curiosity and the desire for new experience. It is, therefore, a sign that surplus energy exists whenever new ideas get a hearing. Nationalism is held to serve two purposes: "to dissipate surplus energy" and "to compensate for the suppression of the social urge and gratify the desire for adventure and achievement in the modern masses." Unfortunately, the arguments in this section are already somewhat out of date, as the 1939 War broke out just after the book was completed. Remembering the stirring scenes of 1914, Creedy states that nations go joyfully to war. But in the autumn of 1939 there was little evidence of joy. Creedy maintains that only a more than adequately fed people with surplus energy can possess an intense nationalism. But in 1940 we see nationalism most rampant in the least well fed of the Great Powers. Nationalism is looked upon as a mad form of primitive religion which has taken the place of decadent Christianity. Communist Russia is seen as internationalistic and so as showing no imperialistic traits!

The function of religion is to make possible the construction of a personality on rational and harmonious principles and to interpose obstacles which will prevent a ready-made personality from being forced upon one by the crowd. Islam, Christianity, and primitive Buddhism are analyzed and similarities are noted, particularly among the two latter. Rhine's work on extra-sensory perception receives favorable notice. At times the author quite forgets his relativistic stand. He vigorously attacks Max Weber who, he says, has "no idea of what religion really aims at." In this same section of the book very brief and rather superficial analyses are made of the arts and sciences.

In Part 3 the psychology of economic life is considered. An extremely radical position is taken which can be indicated here by only an illus-

tration or two. Rather pertinent is his treatment of the American national debt as of July, 1938:

"The figure of $39,000,000,000 has no very important meaning. If we kept a careful record of all the street-car tickets issued throughout the years, we might find that by now they had mounted up to just such a figure. But no one would find this alarming, but merely a rather idle bit of statistics. The Government issue of tickets is on exactly the same basis, merely an instrument of exchange, and these elaborate summations are in fact just as idle in a sane world. In a world which is not sane, they seem to indicate that a certain privileged class is entitled to draw rents (or interests) proportional to this summation, a truly absurd idea." "In a word the economic system of which we are in search will be based on the creation of enough tickets or book entries to render possible noble, worthy, and interesting work for all men."

Although Creedy welcomes the shorter workday, he feels that the demand for this, like the demands for most luxuries, is due largely to the prestige value which short hours carry. Men are said to enjoy work.

In a short chapter on "Sex and the Family" Creedy puts forth the thesis that most of our Occidental sex mores lead to sexual hyperaesthesia. Relative abstinence is necessary so that energy can be diverted to work in the higher arts. Creedy seems more uncertain of his conclusions concerning, and less dogmatic in his social philosophy of, sex than he is of his other topics.

Desperate effort has been made by the author to make his book pedagogically sound. The meat of each chapter is condensed at the beginning, extended with illustrative material throughout the body of the chapter, and then usually briefed again at the end. There is an entire chapter devoted to conclusions. The general reader is advised against the reading of the last or logistic section of the book. Although the reviewer faithfully plowed through these last pages, he confesses to little increased enlightenment and brands himself as a general reader. It appears to him that the labor involved in translating verbal statements of social phenomena into logistic symbols will continue to be largely wasted effort until the material can be presented in truly quantitative form. He rather doubts that the statement, "Sexual desire is not love (of course, this does not mean they cannot be combined)," is made more communicable by expressing it "$F_7) \sim E_7 . \sim E_7) \sim D_7 . ) . F_7) \sim D_7$."

The majority of the 484 pages of *Human nature writ large* will provide pleasant and instructive reading for anyone interested in the social sciences or social philosophy. The book has an intriguing style, and its thesis is earnestly presented. The social psychologist will be particularly interested in its attempts to analyze the stereotypes of Western culture.

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WINKLER, J. K., & BROMBERG, W. Mind explorers. New York: Reynal & Hitchcock, 1939. Pp. 378.

This book is another in that already long list of "popular" presentations of psychology intended for those whose IQ's are limited, either through an oversight of nature or through failure to attend the Iowa

Child Welfare Research Station. The authors, Winkler and Bromberg, are, respectively, newspaperman and physician. According to the book's jacket, Bromberg's M.D. insures the book's scientific accuracy; but this insurance is not always potent enough to curb the literary flights of Winkler, who undoubtedly wrote the book. Winkler's style is breezy and intimate in the best journalistic traditions of the Arthur Brisbane School. His generalities are glittering, his adjectives superlative; and, although accuracy is a matter of small concern, the book makes interesting reading. For the woman who wishes to get enough "psychology" to impress the bridge club or the businessman who wants to confuse the boys on the 8:10 the anecdotal parts of the book should prove useful.

The first two chapters give an account of phrenology and of mesmerism and, on the whole, a fair summary of the place of these scientific culs-de-sac in the history of science. Chapters 4, 5, and 6 deal with medical pioneers in the treatment of the insane and feebleminded: Pinel, Itard, and Charcot in France, Tuke in England, and Todd in this country are the chief figures discussed. Beginning with Chapter 7, the experimental psychologists have their inning under picturesque chapter headings. "The Sportsman-Scientist" is Galton; "A Psychologic Prima Donna" is William James; "A Sire of American Psychology" is G. Stanley Hall; "The Patriarch of Fort Defiance" is Cattell. Terman's work is covered in a chapter entitled "The Measurement of Intelligence." "White Coats and White Mice" sketches the work of Yerkes, Thorndike, Franz, and Lashley. The book ends with chapters on Watson and Freud and with an addendum on the value of, and need for, mental hygiene.

The book contains a number of misstatements, overstatements, inaccurate references, and inaccurate summaries. Some of the errors undoubtedly arise from our authors' ignorance of their material, and some from their incurably hyperbolic style. I quote a few tidbits—gleaned more or less at random—as samples:

Page 143: "Galton," say our authors, "proved that offspring never vary more than one-third from the characteristics of the parent. This principle goes by the imposing title of the *'theory of filial regression.'*" This is a typically loose statement. What the one-third is *of* and what characteristics are covered by this "imposing title" are not stated.

Page 161: Fechner is said, "through laborious testing," to have demonstrated the *"law of psychophysic"*!

Page 182: In addition to his study of the "laws of memory," Ebbinghaus is credited with having trained "the miracle horse, Hans" and "an Arabian steed, Muhamed, to perform calculations like addition, subtraction, and even extracting square roots." Even when written grammatically, this statement is still slightly exaggerated! Apparently, Ebbinghaus was confused with two other fellows.

Page 201: Our authors state that Dewey, Cattell, Sanford, and Jastrow were students "attracted" to Hopkins by G. Stanley Hall. While this is true of Sanford and Jastrow, Cattell and Dewey were already at Hopkins when Hall arrived in 1882.

Page 232: The statement, "H. L. Hollingworth, author of many

volumes on abnormal psychology," would be correct if "abnormal" were omitted.

Page 244: "After exhaustive experiments, involving 250,000 California school children, Terman drew up a list of some 90 tests," etc. Except for the fact that the number of children is about 248,000 too many and that more than half of the tests were taken from Binet's scale, this statement is entirely correct.

Page 253: The expression "primate ape" is redundant, to say the least.

Page 258: Discussing the work of Yerkes, our authors write: "Working as a team, the four Yerkes—mother, father, daughter, son—have pressed constantly forward and have amassed an immense amount of material," etc. Aside from the fact that "the four Yerkes" carries an unfortunate suggestion of vaudeville, I am informed by Dr. Spragg, recently of Yale, that this "team" is more fantasy than fact.

Page 267: The description of the learning of Thorndike's cats given on this page sounds very much as though Thorndike reported "insightful" learning. The implication of the passage is certainly incorrect.

Page 270: Thorndike's honors are many, but he has never been "director of the Division of Psychology at Columbia University." His title is actually Director of the Division of Psychology in the Institute of Educational Research at Teachers College, Columbia.

Page 275: "Every type of intelligence—verbal, mechanical, social, manual, general, arithmetical—has been tested in human beings but no one yet knows what intelligence *is*." We do know, however, that this statement is nonsense!

Pages 280–283: This discussion of Lashley's brain work is jumbled and inaccurate. Lashley did not "conclude that any part of the cortex is able to take over the brightness-discrimination habit in the absence of those occipital areas whose function it normally is." Lashley did find that bilateral destruction of the occipital cortex (including the area striata) permanently abolished visual discrimination of form and pattern. Habits previously acquired were not lost by injury to brain tissue if the destruction was not greater than a critical amount and if the habits were maze, problem box, or brightness-discrimination habits. These habits are lost if the occipital lobes are destroyed and if the habit is one of form or pattern discrimination.

Page 309: It will probably be a surprise (not wholly agreeable) to Professor Yerkes to find that he is one of the workers in Clark Hull's unit at Yale!

This list could be almost indefinitely extended. It would be useful and interesting perhaps, if instructors in general psychology set their students on a still hunt for more errors and misstatements. A copy of the book might be given as a prize to the student finding the fewest errors. In this way the book might serve some useful purpose to professional psychologists; it is hard to see of what value it is otherwise.

HENRY E. GARRETT.

*Columbia University.*

ROBACK, A. A. The psychology of common sense: a diagnosis of modern Philistinism. Cambridge, Mass.: Sci-Art, 1939. Pp. 350.

This definitely is not a book for the psychologist; popular consumption seems to be its objective. Both the title and subtitle are catch phrases which bear little relation to the book's content. Only one chapter—and a mere ten-page one at that—is devoted to a consideration of common sense, and here the treatment is superficial. Throughout the remaining 97% of the book the subject is hardly mentioned. Philistinism is neither diagnosed nor defined; instead, innumerable instances of asininity are cited, ranging from acceptance of Mickey Mouse by the populace to rejection of graphology by the psychologist. The author seems to bemoan the fact that the average person is not above the average.

In the fragment of the book devoted to it, common sense is characterized as follows: "one of the rarest gifts of humanity"; possibly "the missing link between reason and instinct"; more common in women than in men; not equivalent to intelligence; "a *mental set* which enables us to lay out the facts and perceive the relations, at the same time keeping out prejudices and biases, propaganda, and other evil spirits which befuddle the mind"; its essence is objectivity.

The book is an agglomeration of essays comprising the following topics: "What Is Sanity?"; "Sex in Dynamic Psychology"; "The Neurotic Through the Ages"; "The Infantilization of America"; "The 'New' Psychology"; "Technological Fascism and Psychology"; "The Quack in Psychology"; "The Psychology of Success"; "Marriage Lore"; "Graphology as Seen by a Psychologist"; "Music and Moronity"; "Significance and the Condition of Fundamental Preclusion"; "The Concept of Character in a Dictator-ridden World." Roback asserts that "undoubtedly there are many more phases of civilization which could be discussed in a work of this sort, but such a task would require far more than one volume. I naturally chose those subjects which were, to my mind, most important, covering as wide a range as possible, with considerable lacunae, to be sure, that may be filled in later." The reviewer feels that the inclusion of some of the topics, especially "Graphology as Seen by a Psychologist" and "The Quack in Psychology," among the most important phases of civilization shows a certain lack of perspective.

The book cannot be recommended for the serious student of psychology.

WALTER C. SHIPLEY.
*Neuro-Psychiatric Institute of the Hartford Retreat.*

ADVERTISING RESEARCH FOUNDATION. Copy testing. New York: Ronald Press, 1939. Pp. vi+131.

The authors state as their goal the presentation of a ". . . clear and objective analysis of copy testing principles and practices as now applied." One chapter each is devoted to opinion tests, recognition and identification tests, recall tests, inquiry and coupon tests, and sales tests. A final chapter treats sampling. Each chapter on method gives a definition and descrip-

tion, practical applications (including when and where to use, cost, and interpretation of results), warnings, and advantages and disadvantages. The best chapters are those dealing with opinion, recognition and identification, and recall tests. Occasionally there is a lapse in precision of statement, but in spite of this, these chapters present generally accurate accounts of the methods and their uses with adequate criticisms.

This desirable critical attitude is markedly tempered in the chapters on inquiry and coupon tests, and sales tests. Only veiled references are made to many of the uncontrolled variables operating. There is misinterpretation of the meaning of coupon returns, and dismissal of the importance of dilution of replies. It is regrettable that no more than covert reference is made to the largest obstacle in the way of copy testing—that of establishment of criteria. The topic of sampling could be expanded with merit and not relegated to a concluding chapter, and a more detailed analysis of the cost factor would not be wasted.

The value of the book would be enhanced by an appended bibliography, which could list specific references of supporting and conflicting evidence. However, in view of its aim and the audience for which it is intended, this reviewer feels that much has been gained by deleting specific studies and results and inferring only general conclusions.

L. P. GUEST.

*University of Maryland.*

## BOOKS RECEIVED

BURGESS, E. W. (Ed.) *The American Journal of Sociology.* Chicago: Univ. Chicago Press, 1939. Vol. XLV, No. 3.

DURBIN, E. F. M., & BOWLBY, J. Personal aggressiveness and war. New York: Columbia Univ. Press, 1939. Pp. viii+154.

GRABBE, P., in coöperation with G. Murphy. We call it human nature. New York: Harper, 1939. Pp. 120.

LEVINE, A. J. Current psychologies: a critical synthesis. Cambridge, Mass.: Sci-Art, 1940. Pp. 270.

MASON, P. The X of psychology: an essay on the problem of the science of mind. Cambridge, Mass.: Harvard Univ. Press, 1940. Pp. x+216.

RASHEVSKY, N. Advances and applications of mathematical biology. Chicago: Univ. Chicago Press, 1940. Pp. xiii+214.

SHAW, T. L. Art's endurance. Boston: Bruce Humphries, 1939. Pp. 249.

WHEELER, R. H. The science of psychology: an introductory study. (2nd rev. ed.) New York: Crowell, 1940. Pp. xviii+436.

# NOTES AND NEWS

PROFESSOR GODFREY THOMSON has announced that the International Congress of Psychology, which was to have been held in Edinburgh, Scotland, this coming summer, has been postponed because of the war.

THE following additional appointments have been made in the department of psychology at Queens College, Flushing, New York: Dr. Douglas Spencer, assistant professor; Dr. Gregory Razran, instructor; and Dr. Shirley D. S. Spragg, instructor.

THE Consiglio Nazionale Delle Ricerche of Italy has appointed a permanent commission for the applications of psychology. The commission, including several Italian psychologists, is under the chairmanship of Professor Agostino Gemelli.

THE Psychodramatic Institute, a recently established organization for the development of instructors and teachers in psychodrama and its allied subjects, will hold its summer session at Beacon, New York, from June 10 to September 10, 1940. Courses in the technique of the psychodrama, with particular consideration of such problems as educational guidance, marriage counseling, mental disorders, and social maladjustments, will be conducted by J. L. Moreno and staff. Requests for information about the Institute should be made to Psychodramatic Institute, 259 Wolcott Avenue, Beacon, New York.

THE Executive Committee of Recovery, the Association of Former Patients of the Psychiatric Institute of the Illinois Research and Educational Hospitals, announces the completion of the preliminary draft of a new commitment statute, which, after revision, is to be introduced in the next regular session of the Illinois legislature. The main features of the proposed statute are the abolition of court action and the elimination of the "court record," thereby enabling a patient, after proper certification by two physicians, to be admitted to a state hospital without petition, writ, or trial. The Recovery Association, founded in 1937 by 30 former patients of the Institute and now comprising approximately 150 former patients and 500 relatives and friends, publishes *Lost and Found*, a bi-monthly journal in which adjustment problems confronting the recovered patient are discussed. All inquiries should be addressed to Recovery, 1819 West Polk Street, Chicago, Illinois.

DR. GARDNER MURPHY, Department of Psychology, Columbia University, announces that, since many Chinese psychologists have lost all their books, journals, reprints, etc., through bombing, he is undertaking to make a shipment of books and journals to western China early in the summer. He would appreciate receiving as soon as possible any psychological printed matter which American psychologists are able to spare.